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MALARIA

DUREN, A. N. Essai d'étude sur l'importance du paludisme dans la mortalité au Congo Belge. [**Enquiry as to the Importance of Malaria in Relation to Mortality in the Belgian Congo.**] *Ann. Soc. Belge de Méd. Trop.* 1951, Apr. 30, v. 31, No. 2, 129–47. [10 refs.]

The advisability of malaria control in hyperendemic areas was questioned at the International Conference at Kampala [this *Bulletin*, 1951, v. 48, 697] on various grounds, including the opinion that it does not cause any grave harm to the people, and particularly no important mortality. As this question was unresolved and further evidence on the subject was clearly needed the records of research workers, industrial groups, clinics and registration authorities in the Belgian Congo have been searched, and information from them is here presented.

There are many difficulties in interpretation ; the age classification used differs and makes comparison difficult ; certification of cause of death has progressively improved ; figures from hospital autopsies represent the mortality in thoroughly treated groups and are not applicable to the general population. Four sets of data are selected as representative, some adjustment of three of them has been made to ensure comparability of age-groups but not to modify the total mortality. Figures are given below with the addition of a fifth column for comparison showing the death rate from all causes in the same age-groups in England and Wales in 1949.

The death rate per 1,000 living, due to malaria, in the Belgian Congo.

Age	Series				England and Wales all causes 1949
	1	2	3	4	
Under 1 year	40	24·45	31·14	34·2	32
1 year	15·2	9·37	11·8	13	2·5
2 years	10·5	6·4	8·2	9	1·4
Total under 3 years ...	22·2	13·7	17·3	19	8·0

Case fatality is included in the study and also European experience of mortality and case fatality. The latter has sunk progressively from 2·9 per cent. in 1918–20 to 0·48 per cent. in 1941–49 ; in the last period malaria was the cause of 12·3 per cent. of European deaths. The African case fatality is

almost identical with that among Europeans, having decreased from 2·02 per cent. to 0·45 per cent. in the same period. The gross mortality in all age-groups due to malaria is probably 8 per 1,000 per annum. It is concluded that major measures should be taken against malaria in the Belgian Congo.

G. Macdonald

SCHOEMAN, J. J. **Malaria Incidence in South West Africa.** *Pub. Health.* Johannesburg. 1951, July, v. 15, No. 7, 148–51.

South-West Africa is a sparsely populated territory (450,000 sq. miles) with a rather temperate climate. [It lies between the latitudes 17° and 29° south, and west of longitude 21° East except for the Caprivi Strip in the north-east corner, which extends eastwards some 260 miles almost to the Victoria Falls on the Zambesi River.] The southern portion of this vast territory is a flat arid region at an altitude of 3,000 feet with little vegetation and even less water. The western area comprises chiefly the Namib Desert, a waterless waste devoid of vegetation, animal life and insects except for beetles. In the centre of the territory is a well-watered mountainous area with a sub-tropical climate favouring insect life ; Windhoek, the capital, is situated here (23°S.). Stretching east from this mountainous area is bush and plain country with medium rainfall and moderate constant temperatures ; still further east the terrain merges into the great Kalahari Desert. West and north from Windhoek, bush country with medium annual rainfall extends to the Namib desert in the west, and in the north merges into mountainous and plateau country rich in vegetation, which is continued into Portuguese Angola and watered by two great rivers, the Okunene flowing to the Atlantic and the Okovango flowing eastwards to the great Okovango swamp of the Bechuanaland Protectorate. This far northern territory is fairly well populated in the areas known as the Okovango and Ovamboland ; the Kaoko Veld, to the north-west of the Kalahari Desert, is sparsely populated. *Anopheles gambiae* occurs wherever there is sufficient rainfall to maintain breeding places and is in consequence prevalent throughout the northern territory and in the bush lands surrounding Windhoek. It breeds in all types of water, frequently in places " absolutely contrary to the well-known textbook breeding place ". *Anopheles funestus* is common in the well-watered northern territory.

Spleen rates indicate the prevalence of malaria as follows : Windhoek area, 0·5 per cent., Kaoko Veld, 8 per cent., Ovamboland, 50 per cent. and the Okovango, 85 per cent. Parasite indices are comparable. *Plasmodium falciparum* is the common parasite, but both *P. vivax* and *P. malariae* occur. In Ovamboland the " simple tertian " variety is the main infection, but the area is subject to epidemics, whereas in the Okovango malaria is hyperendemic. The farming districts of South-West Africa are comparatively free of malaria, but sporadic outbreaks may flare up.

R. Ford Tredre

LAVERGNE, J. Etude malariologique de l'archipel comorien. [**Malariological Study of the Comoro Archipelago.**] *Rev. Coloniale de Méd. et Chir.* 1950, June 15, v. 22, No. 179, 134–44.

An editorial note states that this report by the Chief of the Madagascar Antimalaria Service was written in 1942. Lack of communications then prevailing between Madagascar and France explains the fact that the report received much less publicity than it merits. It is in fact the first report by an expert malariologist on the malaria conditions of an interesting group of islands.

The Comoro Islands lie in the northern entrance of the Mozambique Channel which separates Madagascar from the African mainland. The group consists of four main islands, Mayotta, Anjouan, Mohilla and Great Comoro, and some small islets of which only two are inhabited. Together the islands cover an area of only 2,147 square kilometres but they support a population of 136,000. The island of Anjouan has a population density of 112 to the square kilometre.

Physiographic and climatologic conditions in the four islands display a remarkable diversity in view of their propinquity. Mayotta, the second largest, is very hilly and has a very indented coastline, but the highest point of the island is very little over 600 metres. Like the north-west coast of Madagascar it has a well marked rainy season from November to April ; the rest of the year is comparatively dry. The maximum temperature is 34°C. in February, the minimum 19°C. in August. Humidity is high throughout the year reaching 90° in the rainy season and never falling below 75°.

Anjouan is slightly smaller than Mayotta but is more densely populated. In the middle of the island a mountain, N'Tingui, reaches a height of 1,500 metres ; in valleys between off-shoots of this mountain are numerous permanent water courses containing abundant clear water. Anjouan has a more temperate climate than Mayotta ; the minimum temperature often falls as low as 16°C. in July. The rainfall is more plentiful ; four or five days of rain are common in both July and August, the driest months of the year. In spite of this the humidity is lower than in Mayotta.

Mohilla, the smallest island of the four, has the luxuriant vegetation that characterizes Mayotta, and numerous abundant watercourses as has Anjouan. Mountains run from north-west to south-east ; the highest are between 800 and 900 metres. The climate resembles that of Anjouan but is not so agreeable.

Great Comoro is much the largest of the four islands, 1,145 square kilometres, with a population of 75,000. It is very different from the other islands : it is an enormous block of volcanic rock and lava with an active volcano, Karthala, 2,400 metres high. There is no permanent water course ; rain infiltrates immediately into the fissured soil. There are two springs with a very limited outflow but the water disappears into the soil again within a short distance. Enormous ravines carry storm water to the sea ; a simple storm may produce a raging torrent in such a ravine, but a day without rain dries it completely. The volcano Karthala, almost always cloud-capped, ensures abundant rainfall at all seasons of the year : only the extreme north of the island, which is furthest away from the mountain mass, has a real dry season. The southern part of the island with its tropical vegetation, has always abundant rain. Tropical vegetation ceases at a height of 350 metres : from 1,000 to 1,500 metres there is forest : higher still there is a belt of brush and scrub : above 2,000 metres there is the cone of volcanic mud and cinders. The population is concentrated near the shore ; villages are rarely higher than 500 metres. The mean temperature is remarkably constant, only 5° difference between the mean temperatures of January, February, the hottest months, and July, August, the coldest.

The malaria survey of the islands was made in June and July, the most unfavourable season of the year from the point of view of anophelism. At Anjouan there was a suspension of anopheline breeding and malaria transmission : at Mayotta, Mohilla and the southern part of Great Comoro there was only a slowing of anopheline breeding and transmission : in the northern arid part of Great Comoro there was extremely little breeding or transmission.

Anopheles gambiae appeared to be the most important transmitter. *A. funestus* was less prevalent. *A. funestus* var. *imerinensis* was very rare. *A. squamosus* and *A. mauritianus* were also found. *A. gambiae* var. *melas* [*A. melas*] was not found. There was nothing unusual about the breeding places of these

anophelines except in Great Comoro : here during seven or eight months of the year there are no natural breeding places and the anophelines breed in open water tanks which are characteristic of the native villages. This domestication of these anopheline species is a remarkable phenomenon.

Malaria endemicity in Great Comoro varies greatly from village to village, being dependent upon the number of water tanks and the time elapsed since the importation of infection. Thus at Moroni there was a spleen index of 65 per cent. (550 children examined) and a parasite index of 17·5 per cent. At Foumbouni the spleen index was only 7 per cent. but the parasite index was 23·5 per cent. In the latter place infection was recent and there was little or no premunition. Dispensary returns show that there is little if any seasonal variation of malaria transmission in the southern part of Great Comoro.

In Mayotta spleen rates varied from 40 to 90 per cent. and parasite rates from 10 to 18 per cent. Here there is evidence of long-standing hyperendemicity and very considerable premunition. Severe epidemics do not occur. There is a great preponderance of *P. falciparum* infections. There is no natural increase of population.

Malaria conditions in Mohilla are similar to those in Mayotta but of greater severity : almost all the population are infected. Birth rates are low and the population decreasing. The proportion of malaria cases to total dispensary attendances varies little from month to month.

Anjouan is the least malarious of the four islands. The spleen rates vary from 20 to 55 per cent. but very large spleens are not found. The average parasite rate was only 15 per cent. Though *P. falciparum* is preponderant along the coast *P. vivax* is more prevalent on the high land. The high lands are indeed remarkably healthy.

With regard to control measures, the author would rely on suppressive medication except in Great Comoro where the breeding of anophelines in water tanks might be prevented or controlled without much difficulty. [The editorial note reminds us that the report was written in 1942. Today the author would almost certainly have considered the possibility of control with DDT.]

Norman White

ABHAYARATNE, O. E. R. **The Influence of Malaria on Infant Mortality in Ceylon.** *Ceylon J. Med. Sci.* (Sect. D.) 1950, June, v. 7, Pt. 2, 33–54, 5 figs.

The recorded infant mortality rate in Ceylon up to 1946 was always high, even by the standards of that half of the world in which a high rate is normal, and records since 1900 are believed to be accurate. The mean rates for the first four decennia of this century per 1,000 live births were 180, 196, 183 and 171. Examination shows very marked fluctuations within the ten-year periods. There has been a total of ten peaks, seven of which coincided with known malaria epidemics, two with influenza epidemics, and one, in 1909, is unexplained.

When the figures are analysed on a regional basis, and compared with the known endemicity of malaria which corresponds to climatic divisions, a marked correlation is found. Data are given for the periods 1916 to 1925 and 1926 to 1939. They show high rates (242 and 200) in the highly endemic dry zone, much lower rates (132 and 118) in the non-malarious wet zone, and intermediate rates in registration districts which include parts of the intermediate as well as of the wet or dry zones. Epidemic fluctuations are much greater in such districts than elsewhere.

The difference noted applies not only to the rate as a whole, but to the mortality under one week and under one month of age, for which data are given. [A deleterious effect of malaria on the health of the mother is presumably indicated.] *G. Macdonald*

BOLIVIA : MINISTERIO DE HIGIENE Y SALUBRIDAD. División de Endemias Rurales. Historico del servicio antipaludico en Bolivia y trabajos realizados hasta fines del año 1950 [MOSCOSO CARRASCO, C., Jefe del Servicio]. [**History of the Antimalaria Service in Bolivia and Activities up to the End of 1950.**] 57 pp., 4 charts & 2 maps. 1951. Cochabamba-Bolivia.

JASWANT SINGH & NAIR, C. P. **Abnormal Forms of *Plasmodium vivax* Grassi and Feletti, 1890.** *Indian J. Malariology.* 1950, June, v. 4, No. 2, 193–202, 1 chart & 2 pls. [29 refs.]

A description is given of the occurrence in blood-smears of a 7-month-old child in Delhi of a large proportion (96 per cent.) of abnormal malaria parasites in an infection containing 4 per cent. typical *Plasmodium vivax*. Such parasites were present both before paludrine [proguanil] was administered and 24 hours after treatment.

In blood films stained by the J.S.B. method, the infected erythrocytes were enlarged and stippled with Schüffner's dots, their average diameter being about 8·3 μ, *i.e.*, less than in the typical *vivax* forms present (about 9·3 μ). The erythrocytes also showed certain features of *P. ovale*, 9 per cent. being oval in shape, and 5 per cent. with fimbriated ends.

All the abnormal parasites were rounded and solid in appearance, most of them having the characters of trophozoites occupying $\frac{1}{3}$–$\frac{2}{3}$ of the host-cell, with insufficiently defined gametocytes taking up $\frac{2}{3}$–$\frac{3}{4}$ of the host-cell. A few small forms were in binary fission, but later stages of schizogony were absent. There were also 4 per cent. of band-forms, and chromatin was abundant in all the parasites.

In general, the abnormal parasites showed some resemblance to *P. ovale*, and were similar in this respect to a number of cases reported in the literature, in which such forms were found in strains of *P. vivax*.

Among the peculiarities of this infection is the large number of parasites extruded from the host cell, and the presence of giant forms of residual red corpuscles partially or completely enclosing the parasite.

In considering the possible causes of these abnormalities, the authors have excluded medicinal treatment, since in the first instance the blood films were made before treatment. There could also be no question of faulty technique, or the influence of the stain used (J.S.B.), for similar forms appeared in films stained by the traditional Giemsa method. However, they admit the possibility that undernourishment of the patient—with the resulting vitamin deficiency—might have been responsible for the appearance of abnormal parasites.

The paper is illustrated by two plates depicting the parasites.

C. A. Hoare

EICHLER, W. Anopheles-Beobachtungen in Südrussland. [**Observations on *Anopheles* of Southern Russia.**] *Riv. di Malariologia.* 1951, Feb., v. 30, No. 1, 29–38, 5 figs. [20 refs.] English summary.

This paper reports the presence of *Anopheles maculipennis messeae*, *A. m. typicus*, *A. m. atroparvus* (at Ssolênij on the Taman peninsula only) and *A. hyrcanus*, in the Kuban district (Black Sea) of south Russia. *A. m. messeae* is the predominant mosquito, breeding in large numbers in the reedy swamps of the low-lying river plains. Its eggs differ from those of European *messeae*

but adequate details are not given in this paper. Adult collections in the village of Achtanisowskaja demonstrated that the mosquitoes occurred in large numbers in stables and, as regards houses, chiefly in those on the edges of the village. The number of mosquitoes per house varied from nil to 93 ; bedrooms were the usual rooms frequented.

Several summer generations, and overwintering females, are reported for *typicus*, the mosquito prevalent in the hilly regions. In the Taman peninsula, the breeding places of *messeae* may dry up in summer and those of *atroparvus* in Lake Janowskowo may become excessively brackish after the spring floods of the rivers have subsided. It is stated that under these conditions, oviposition ceases, a phenomenon to which the author applies the term " summer phase ". The author, although implying that the conditions of the breeding places may initiate this state, concedes that the summer temperatures may also be an important factor. [The paper does not provide convincing evidence that oviposition does, in fact, cease in these areas where breeding places dry up or become otherwise unsuitable for breeding.]

Short lists of plants are given for the swamps and channels characteristic of the river plains.

The illustrations consist of three photographs of breeding places and one of different patterns of *messeae* eggs from females taken in the area. The paper is concerned with observations made in 1943. *D. S. Bertram*

EICHLER, W., LÖPMANN, A. & PAGAST, F. Beobachtungen über *Anopheles hyrcanus*. [**Observations on** *Anopheles hyrcanus*.] *Ztschr. f. Tropenmed. u. Parasit.* Stuttgart. 1951, June, v. 3, No. 1, 25–32, 3 figs.

These observations on *Anopheles hyrcanus* were made in Yugoslavia, Ukraine and Kuban (Caucasus) during 1942 and 1943. The form found in Yugoslavia was identified as the sub-species *pseudopictus* but the status of the forms found in the other areas is uncertain.

The eggs are described and figured ; the larvae are said to rest at the water surface with their heads down, almost like those of an *Aëdes* mosquito, while the adults rest on a vertical surface with their abdomens projecting almost horizontally.

Females bite outside, most commonly among reeds in and around their breeding places, though they have been observed biting indoors. Males swarm at sunset.

For the following reasons *A. hyrcanus* seems to be less important as a vector of malaria than *A. maculipennis* : malaria incidence is highest in areas where only *maculipennis* [? *atroparvus*] is found and lowest where the mosquitoes are mostly *hyrcanus*. *A. hyrcanus* is rarely found in houses where malaria occurs and has a shorter flight range than *A. maculipennis*.

H. S. Leeson

QURAISHI, M. S., AHMED, M. & GRAMICCIA, G. **Pre-Monsoon Malaria Transmission in the District of Mymensingh, East Pakistan.** *Bull. World Health Organization.* Geneva. 1951, v. 3, No. 4, 673–82.

The area concerned in this report is in the higher plains of East Bengal which are, however, only some 200 feet above sea-level. Here *Anopheles philippinensis* is the malaria vector ; tanks with submerged vegetation are the chief breeding places of this species. This was the finding contained in a report by IYENGAR [this *Bulletin*, 1945, v. 42, 625] who further stated that September–November is the main malaria transmission season : the earlier May–June equally high peak in the malaria incidence curve was attributed by

him to relapses. This question of pre-monsoon, April–June transmission of malaria has been further studied by the authors of the present paper. They find that at this season marshes and dead rivers (branches of rivers that have been cut off from the main stream but which contain perennial water) constitute important breeding grounds for *A. philippinensis*. There is a positive correlation between the proximity of a village to marshes or dead rivers and a high spleen index of its population and a high incidence of the vector species. One *A. philippinensis* with sporozoite infection was found in May and two in June, all three in villages near marshes. Between April and July ten infants under five months of age were found infected with malaria out of 61 examined. Only one of the infected infants came from a village not near a marsh. Lower spleen and parasite rates were found in villages that had been sprayed with DDT in April than in villages not sprayed until June and July.

Norman White

McArthur, J. **The Importance of** *Anopheles leucosphyrus*. *Trans. Roy. Soc. Trop. Med. & Hyg.* 1951, June, v. 44, No. 6, 683–94, 1 map. [73 refs.]

This paper is a review of the progress of knowledge regarding the relation of *Anopheles leucosphyrus* to malaria in the Far East [see this *Bulletin*, 1947, v. 44, 788].

A. leucosphyrus is a jungle-breeding mosquito which tends to disappear with the removal of shade and the admission of sunlight into its breeding places. It is a variable species, and, in its different forms, has now been identified across Asia, from India to Celebes, and from Java to Formosa. It is not uncommon in many parts, but is sometimes difficult to find even when present in large numbers. It has generally been considered of little importance as a vector of malaria, on account of its apparent rarity.

This species was first suspected of malaria transmission in 1914, in North Borneo, and since then has been recorded as suspect, on epidemiological evidence, in Sarawak, Indo-China, Burma, Assam and elsewhere. The first positive dissection of *A. leucosphyrus* for malaria parasites were made in 1918 in Sumatra, and subsequently both a strong preference for human blood (Netherlands Indies, 1932) [this *Bulletin*, 1932, v. 29, 830] and definite salivary gland infections (Assam, 1938) [*ibid.*, 1942, v. 39, 177] have been demonstrated.

A. leucosphyrus has been found to be more common than was previously thought to be the case, as for instance in the forest-covered plains of Indo-China, where, in the rainy season, it is the most common anopheline. It has been found, also, that wherever an adequate number (40 or more) of dissections have been made, malarial infection has been proved.

In Assam, it is now regarded as a most significant complementary species to *A. minimus*, being responsible for most of the malaria transmission during the rainy season and at high altitudes ; and in North Borneo, breeding in jungle-covered ravines and overgrown streams, it is the most important vector. Clearing of such vegetation in and around towns has eliminated the mosquito from those areas. In Malaya, *A. leucosphyrus* has never been considered a vector, but the records of dissections are very few.

Of the different forms of *A. leucosphyrus*, the evidence is in favour of the type form being the one of importance as a malaria vector, the only exception being var. *hackeri* in Celebes.

The data are put into the form of a map showing the geographical distribution of positive dissections, and also into a table giving details of all dissections of *A. leucosphyrus* recorded in the literature.

The author considers that this species is more important as a vector of malaria in Asia than has hitherto been appreciated. *A. J. P. Goodchild*

RACHOU, R. G. Observações sôbre o ciclo evolutivo dos anofelinos do subgênero *Kerteszia* em laboratório : fase de ovo. [**Notes on the Life Cycle of** *Kerteszia* **in the Laboratory : the Egg Stage.**] *Rev. Brasileira Malariologia.* Rio de Janeiro. 1950, Apr. 30, v. 2, No. 2, 111–18. English summary.

Working at Florianopolis in Southern Brazil, the author made observations on three anophelines of the subgenus *Kerteszia* (breeding doubtless in the axils of Bromeliads). In *K. cruzii, homunculus* and *bellator*, about 70 eggs are deposited per oviposition. The duration of the egg stage is remarkably variable, *e.g.*, 2 to 18 days in *cruzii*, and 4 to 12 in *bellator* ; it seems that prolongation of this stage is not due simply to low temperature.

P. A. Buxton

FUHRMANN, G. & HARTEN, R. Der Serum-Eiweissspiegel und die Serum-Labilitätsreaktionen bei der Malaria Tertiana. [**Serum Protein and Serum Lability Reactions in Benign Tertian Malaria.**] *Ztschr. f. Tropenmed. u. Parasit.* Stuttgart. 1951, Apr., v. 2, No. 4, 457–71. [22 refs.]

This paper contains a detailed analysis of various serum lability reactions including the Takata, Weltmann and thymol turbidity tests, together with measurement of serum protein fractions in *vivax* malaria (recent infections ; relapsing cases with up to 4 relapses ; relapsing cases with more than 4 relapses).

The authors conclude that the serum protein reactions indicate both acute inflammatory processes and hepatic damage. The hepatic element predominates in recent infections ; there is no evidence of permanent liver damage.

B. G. Maegraith

MEYTHALER, F. & SCHAIBLE, E. Über die positive Wassermann-Reaktion bei Malaria. [**Positive Wassermann Reactions in Malaria.**] *Ztschr. f. Tropenmed. u. Parasit.* Stuttgart. 1951, June, v. 3, No. 1, 4–17. [55 refs.]

That the Wassermann test is not strictly specific for syphilis was recognized by Wassermann himself when he spoke of " group specificity ". It is known now that many diseases injuring tissues and organs rich in lipoids may produce a positive test.

Literature is reviewed for results obtained by Wassermann and related tests in malaria.

Hardly any author doubts that malaria may influence the Wassermann test, yet as to degree and percentage opinions differ widely. This uncertainty is due to many elements : kind of antigen used ; haemolytic substances during fever periods ; phases of infection ; parasite findings ; differences of plasmodial strains and their faculty to incite antibody production ; racial and seasonal influences. A positive cerebrospinal fluid test is unanimously denied. Cases of relapsing malaria seem to give more negative or weakly positive tests, yet the prognostic value of such results is doubted. Tests do not differ for genuine and induced malaria. The Wassermann test is more often positive in malaria than are flocculation tests. Most authors agree that factors turning the tests positive are more transitory in malaria than in syphilis. It is furthermore remarkable that the various antigens used do not follow strict rules. Cholesterinized extracts may yield stronger reactions than ordinary extracts, but not always. Such contradictions are typical for the results published.

Specific antibodies against plasmodia were found by many authors independently of each other and by different methods.

Parallelism of the serum test and blood sedimentation rate could not be proved. A rise of bilirubin level in the blood was noted during acute fever only or shortly after.

B. G. Maegraith

VAN DEN BERGHE, L. & CHARDOME, M. **An Easier and more Accurate Diagnosis of Malaria and Filariasis through the use of the Skin Scarification Smear.** *Amer. J. Trop. Med.* 1951, July, v. 31, No. 4, 411–13.

Malaria and filaria infections can be very accurately diagnosed by the examination of fluid obtained from scarification of the skin. A site in the middle of the scapular region is chosen, the skin is superficially cut so that little bleeding takes place, and finally the scarified area is squeezed between the thumbs and forefingers. The dermal capillary blood thus obtained is spread either as a thick or thin film on a slide and is stained and examined in the usual way.

The microfilariae of *Mansonella ozzardi*, *Acanthocheilonema perstans*, *Dipetalonema streptocerca*, *Wuchereria bancrofti*, *W. malayi* and *Onchocerca volvulus* are readily found and diagnosed by this method. Also, the four species of malaria parasites are easily seen in such smears both as schizonts and gametocytes.

The authors claim that a higher percentage of malaria infections is revealed by this method than by the examination of thick films ; in highly endemic areas, the scarification method gave a parasite rate of 98 or 99 per cent. while thick films showed 90 per cent. or less. In chronic infections the parasite is much more easily seen in the dermal than in the ordinary blood film, and in several thousand cases there was a greater frequency of mature schizonts and of gametocytes in the skin.

[The importance of these observations need hardly be emphasized—if confirmed they will inevitably have a profound effect on (*a*) methods of routine diagnosis of malaria, (*b*) parasite surveys, and (*c*) estimations of the carrier (*i.e.*, gametocyte) capacity of individuals. No details are given in the present paper though some comparative figures were provided in the original observations of PEEL and VAN HOOF, see this *Bulletin*, 1949, v. 46, 7.]

P. C. C. Garnham

KHAN, N. **Cerebral Malaria.** *Indian Med. Gaz.* 1951, Mar., v. 86, No. 3, 91–4.

The author describes the method of treatment of cerebral malaria used by him at the Tata Hospital, Jamshedpur, Bihar. As soon as the diagnosis is made an intramuscular injection of 0·25 gm. methyl-sulphonate-mepacrine is given. This is repeated every six hours until the patient recovers consciousness and can swallow. Concurrently from 50 to 200 mgm. of nicotinic acid with 20 to 30 cc. of 25 per cent. glucose is given intravenously, extremely slowly : this intravenous injection is repeated every hour or two hours until consciousness is regained. The object of the nicotinic acid administration is to produce vasodilatation of the brain. Intranasal intragastric feeds are given every two hours. As soon as the patient is able to swallow 0·6 gm. of mepacrine is given daily for seven days.

During two years in which this method of treatment was adopted deaths from cerebral malaria in this hospital numbered 30 and 14 per thousand of admissions for *P. falciparum* malaria, as compared with 95 and 98·2 per thousand in two previous years when cases of cerebral malaria were treated with intramuscular injections of quinine.

The pathological and clinical features of cerebral malaria are discussed and clinical details are given of three cases.

Norman White

JASWANT SINGH. **Antimalarial Drugs.** *Indian J. Malariology.* 1950, June, v. 4, No. 2, 185–8, 1 folding table. [Bibliography.]

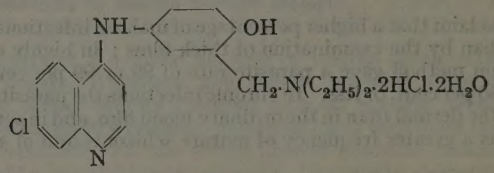
The author has rendered a valuable service to his fellow workers in India and elsewhere by producing in tabular form the characteristics of the numerous

antimalarial drugs which in recent years have proved their worth in the treatment and suppression of malaria. The drugs are classified into quinoline, acridine, 4-aminoquinoline, 8-aminoquinoline and biguanide series. The chemical structure of each drug is illustrated, the phase of development of the parasite against which the drug is operative is indicated, and notes regarding toxicity and dosage in differing circumstances contain all that is essential.

Norman White

BERTAGNA, P. Aperçu général de la documentation relative à l'amodiaquine. [**Survey of the Literature on Amodiaquine.**] *Bull. World Health Organization.* Geneva. 1951, v. 4, No. 2, 267–81, 1 fig. [27 refs.]

The substance named in the title, also known by the synonyms Camoquine, Cam-aqi, Miaquine, SN 10,751, was synthesized in America during the second World War by Parke Davis. It is 7-chloro-4-(3′-diethylaminomethyl-4-′-hydroxyanilino)-quinoline with the following structural formula :—



and has been described in the "Survey on antimalarial drugs 1941–1945" edited by WISELOGLE [this *Bulletin*, 1947, v. 44, 1106] ; two early reports on its activity were given [*ibid.*, 1947, v. 44, 698 ; 1948, v. 45, 39] and have been followed by many others. The substance is a yellow crystalline powder of m.p. 208°C. and is soluble to the extent of 1 gm. in 5 ml. water at 25°C. It is generally supplied in tablets containing 0·2 gm. of the bihydrochloride, and is administered preferably by mouth. It is rapidly absorbed from the gastro-intestinal tract and becomes distributed in tissues, especially the liver, at a level higher than that in plasma. There was also some localization in the non-diffusible elements of the latter, and the drug has been detected in leucocytes 23 days after the end of treatment. In the animal body it appears to be partly metabolized with relatively slow elimination of the products formed. The substance has been widely tested in avian and mammalian malarial infections and its toxicity determined for many laboratory animals. Resistance to the drug could not be produced in *P. lophurae*. It possessed marked suppressive but not prophylactic properties in several species of bird malaria. In man it has been proved active against the asexual forms of plasmodia and against the sexual forms of *P. vivax*, but had little or no effect against those of *P. falciparum*. It is a good suppressant and possesses marked schizonticidal properties. Fever usually disappeared after a day or two and parasites after a period of 1 to 4 days. Relapses do occur but generally after a longer period than with other drugs. The drug is given preferably in a single dose by mouth, as follows :—

Adults 0·6 gm.
Children 5–15 years 0·4 gm.
Children under 5 years 0·2 gm. or less.

or in several doses on the basis of 10 mgm. per kilo., which appears to be the minimal amount for satisfactory treatment. Any signs of toxicity noted in man have been of transient character. The drug is not contraindicated in pregnancy or in cases of liver cirrhosis.

[References are given to many papers on the use of this substance and considerable details about dosage and toxicity which should be consulted in the original.]

J. D. Fulton

BANERJIE, N. R. **Acute Abdominal Symptoms with Paludrine.** *J. Indian Med. Ass.* 1951, Aug., v. 20, No. 11, 415–16.

The author, writing from Gorakhpur, records 5 cases in which acute abdominal manifestations, simulating appendicitis in the worst cases, occurred after the exhibition of paludrine [proguanil] in the treatment of malaria. In one case, the symptoms appeared 15 minutes after the second of two doses each of 100 mgm. of paludrine (with 5 hours between them). In another, the pain and vomiting arose "after 3 tablets of paludrine". In each of these cases, paludrine was given to treat relapses of the benign tertian malaria a few months later and "the same disaster followed". The patients responded to other antimalarial drugs, apparently without mishap. In the other 3 cases (two of *P. vivax* infection and one without proved parasitaemia) the dosage of paludrine and the intervals are not stated, but the same after-symptoms appear to have occurred.

The author does not attempt to explain the phenomena [and the evidence adduced is not sufficient to allow of an unequivocal judgment on the relation of the manifestations in these cases to paludrine as such].

H. J. O'D. Burke-Gaffney

RUIZ SANCHEZ, F., NIEVES, M., QUEZADA, M., PAREDES, M. & RIEBELING, Rebecca. El tratamiento del paludismo con aureomicina. [**The Treatment of Malaria with Aureomycin.**] *Medicina.* Mexico. 1951, May 25, v. 31, No. 628, 183–8, 9 figs.

A man aged 27 suffering from fever was admitted to hospital. Investigation showed that he was suffering from a *Brucella melitensis* infection ; at the same time his blood contained numerous *P. vivax* parasites in different stages of evolution. He was given aureomycin, 8 mgm. per kgm. of body weight a day for 11 days. The temperature fell below normal after two days of treatment. Subsequent antimalarial treatment was not necessary ; the parasites had disappeared. This experience prompted the trials of aureomycin in the treatment of malaria which are reported. Nine patients with naturally acquired malaria, six *P. falciparum* and three *P. vivax* infections, were given aureomycin, in doses from 30 to 34 mgm. per kgm. body weight a day, for 6 to 8 days. The average duration of fever after the beginning of treatment was 4 days. *P. vivax* parasites disappeared from the blood on the 6th or 7th day as did the asexual forms of *P. falciparum.* The drug had no action on *P. falciparum* gametocytes.

Seven patients undergoing malaria therapy, who had been inoculated with blood containing *P. vivax*, were similarly treated. One of these received intravenous injections of aureomycin, 20 mgm. per kgm. body weight daily, for 8 days. In these cases fever was suppressed in 3 to 5 days, parasites in 3 to 7 days.

Norman White

NOYAN, A. **Malaria Prophylaxis with Atebrin.** *Acta Med. Turcica.* 1950, Apr., v. 2, Nos. 1/2, 1–8.

The author, who is a Professor of Medicine in Ankara University, discusses briefly the general principles underlying the prevention of malaria and the drugs and their schedules which have been employed in chemoprophylaxis.

After a discussion on the relative merits of different schedules of atebrin [mepacrine], he suggests that the use of 0·3 gm. daily for 5 successive days at 2-week intervals is economical, effective, practicable and less liable to produce cumulative and toxic effects than some of the better-known schedules.

In 1946, this schedule was carried out in a number of military establishments in Turkey and was compared with the concurrent trial of 0·3 gm. given twice

a week. In the first group, a total of 6,077 persons received the daily schedule and in the second, 7,872 received the bi-weekly schedule. The number of cases of malaria recorded were 880 (14 per cent.) and 1,002 (12 per cent.) respectively.

These trials covered officers, students and other ranks in military units, military colleges and schools in a number of different areas.

In the towns of Konya and Mersin, about 150 soldiers did not benefit from the prophylaxis and the relapse rate in them was from 35 to 53 per cent. It is stated that these troops had possibly been re-infected repeatedly as they had been stationed in highly malarious regions : so that the result " is not due to ineffectiveness of the drug ".

The author emphasizes that it is not possible to assess the value of this method from the results of one or two years' trial and that a large-scale statistical survey would be preferable.

The results are set out in two tables [the totals in Table II are added up incorrectly, but this is possibly due to misprinting as the final percentages are not affected]. *H. J. O'D. Burke-Gaffney*

CHAUDHURI, R. N. **Suppressive Treatment of Malaria,** with Statistical Analysis by S. J. POTI. *Indian J. Malariology.* 1950, June, v. 4, No. 2, 115-33, 4 figs. [27 refs.]

This report relates to an inquiry in a rural area of West Bengal as to the relative values of paludrine [proguanil], chloroquine and camoquin in the suppression of malaria. A preliminary trial with paludrine was carried out in 1947 in Katalgarh, a small village in the Hooghly District with a population of about 300. There are numerous bamboo groves and water tanks and rice fields all round the village. Malaria is very prevalent during the rains ; the maximum incidence is in October and November. In September 1947, 83 children were examined : 77 had enlarged spleens (57 per cent.) and 24 had parasites in the blood (29 per cent.) : 19 of these 24 had *P. falciparum* infections. Paludrine was given to 60 of the poorest inhabitants, the remainder of the population serving as a control. Spleen and parasite rates of children of the treated group were 72 and 50 per cent. as compared with 47 and 16 per cent. of children of the control group. Each person treated received a tablet of paludrine (0·1 gm.) each Sunday and Wednesday from September 11 till the end of December. Children under 4 received half a tablet each. At the end of the experiment spleen and parasite rates of the paludrine group were 45 and 17 per cent. ; of the control group 46 and 22 per cent. There was a ten-day interruption in the treatment in October, the height of the malaria season.

For the main inquiry the work was extended to 8 villages in the same district. The combined population was divided into five groups each of about 250 persons. The members of these groups received treatment as follows : (1) paludrine 0·1 gm. twice weekly, (2) paludrine 0·3 gm. once weekly, (3) camoquin 0·2 gm. once weekly, (4) chloroquine 0·25 gm. once weekly, (5) the control group received quinine in case of fever only. Reduced doses were given to children under 10. Drug administration started in July 1948 and continued to December. The reduction in parasite rates was significant in all treated groups, especially at the height of the transmission season. Treatment 2, paludrine once a week, was the least effective in this respect. Chloroquine and camoquin were very effective in preventing and in reducing splenic enlargement : in this respect also paludrine once weekly was the least effective suppressive. Febrile attacks of malaria were rare among persons taking drugs regularly ; many in the control group suffered from repeated attacks. All the drugs were well tolerated ; children and pregnant women showed no intolerance.

Suppressive therapy has a valuable rôle to play in rural areas wherever anopheline control is impracticable. *Norman White*

RAFI, S. M. & SHAH, I. A. **Paludrine as a Causal Prophylactic in Hyperendemic Areas.** *Pakistan J. Health.* 1951, Apr., v. 1, No. 1, 42–6. [10 refs.]

Fifty-seven adults and 30 children living in the village of Tathi Sialan in the Sheikhupura District of the Punjab were placed on proguanil prophylaxis from August 1 to December 31, 1949, the period of maximum malaria transmission in that area. Two régimes were under trial, adults receiving either 300 mgm. proguanil in a single dose once weekly or 100 mgm. on separate days twice weekly, the majority being on the former schedule. Children were given proportionately smaller doses according to age. Spleen and blood surveys were carried out immediately before commencement of drug administration and 14 days after its cessation. Parallel observations were made on 50 adults and 38 children living in another village in the same district under comparable conditions, except that no prophylactic drug was given.

Seventeen adults and 13 children experienced attacks of fever while under proguanil prophylaxis, but in only 3 cases were parasites found in blood smears. These were all *P. vivax*. During the same period there were 49 cases of proved malaria (31 *vivax*, 17 *falciparum* and one mixed infection) among those under observation in the comparison village. The spleen rate among those under proguanil prophylaxis fell from 42·5 to 3·4 per cent. and the parasite rate from 37·9 to 5·8 per cent., while in the comparison village both these rates were higher at the end of the malaria season than at the beginning. The authors consider that both the régimes employed for prophylaxis were equally effective. *G. Covell*

SABATINI, A. La lotta antianofelica nell'Agro Romano negli anni 1947–1950. [**The Campaign against Anopheles in the Agro Romano during 1947–50.**] *Riv. di Parassit.* Rome. 1951, July, v. 12, No. 3, 185–92, 2 graphs. English summary.

The Agro Romano is the rural country which surrounds Rome and which includes the river Tiber between the city and the sea coast, where it involves 40 kilometres of coast, the latter being 45 kilometres from the city. For many years before the war malaria control in this area had been carried out by means of drainage and other reclamation works and by persevering larvicidal measures. [The Malaria Institute in Rome took the leading part in these measures, and at one time the Health Division of the Rockefeller Foundation shared in this work.] Apart from some rise in 1939–1940, the number of notified cases of malaria had become stabilized around 1,000 a year until 1944 and 1945 when the notified incidence, under war and invasion conditions, went up to 9,063 and 11,025 cases respectively. Then a curative and preventive drive, including some DDT treatment, had a dramatic result, notifications dropping to 2,500 for 1946.

From 1947 on, a carefully planned organization has regularly applied DDT spraying against the adult insect in this area with the exception of the immediate surroundings of the city and of the northern sector of the area, which are subject to little or no malaria. Each of the other 5 sectors has been provided with a spraying squad of 8 men and one foreman. All the spraying solution of DDT, to which chlordan has been added since 1949 to deal with DDT-resistant house-flies, is prepared in a central depot in Rome. For the two inland sectors the squads are based in this central depot, while the 3 coastal sectors each have their own branch depots from which the local squads draw their supplies. The spraying equipment is standardized and a staff of five men

at the central depot are responsible for all repairs, as well as for the preparation of the insecticide mixture. A field entomologist, assisted by two inspectors, is in charge of the organization. Since 1950, bicycles have been substituted for motor transport and have resulted in better individual mobility with higher efficiency. In the light of experience, the insecticide treatment has been discontinued in some areas such as the urbanized Lido on the coast, while spraying has been extended to some parts of the northern sector. Close on 100,000 structures in all, entailing nearly 5 million square metres of surface, are sprayed each year.

This control of adult mosquitoes, and of other insects, by means of residual insecticide spraying has given impressive results : 435 cases of malaria have been notified in 1947, 135 in 1948, 16 in 1949 and none in 1950. The number of *Anopheles* collected from catching stations has rapidly dropped to nil in 1950, and it is stated that adult catches had persisted at a high figure in the years before the war in spite of all the reclamation and larvicidal measures then carried out.

J. Cauchi

MECHALI, M. D. La lutte anti-paludique au Maroc. [**Antimalaria Campaign in Morocco.**] *Maroc Méd.* 1950, Jan., v. 29, No. 296, 112–17, 6 figs.

This article, in a special number of *Maroc-Médical* devoted to Public Health, describes the organization of the existing antimalaria campaign in Morocco, the general policy being followed, and the results already obtained. The central administration is attached to the Institute of Hygiene. It has two laboratories, one for haematology and one for entomology ; a research centre situated in a hyperendemic malarial area, and two antimalaria sections to carry out inquiries and to reinforce regional services in times of epidemic emergencies. At the regional level, malaria control is one of the responsibilities of the local Health Administration which has for this purpose one to five antimalaria sections, according to the intensity of malaria in the region. In the conditions prevailing in Morocco suppressive medication cannot be expected to secure any great measure of control and measures against the mosquito vector now form the basis of antimalarial work.

Collaboration with irrigation engineers, supervision of irrigation and technical advice concerning details of reclamation projects do much to eliminate or diminish mosquito hazards. For the protection of towns or large rural centres the suppression or neutralization of all mosquito breeding places within a distance of 5 kilometres of the inhabited area is the ideal procedure. At present, 29 places are protected by this method in Morocco. In rural areas measures against the adult mosquito are more practicable. In Morocco the most important vector, *A. maculipennis*, is a domestic species. If the population is stable good results are achieved. For scattered dwellings, difficult of access, even this method of control becomes too costly. In such cases, frequent in Morocco, recourse must be had to suppressive medication. This measure is generally confined to children, the aim being to enable them to develop sufficient premunition with lessened risk. The weekly distribution of a synthetic suppressant is the method employed. Finally epidemic outbreaks of malaria which occur from time to time call for energetic mass treatment.

Today all large centres of population, comprising more than a fifth of the total population of the country, are free from malaria thanks to antilarval measures. Infant death rates in endemic areas have decreased in recent years. Spleen rates in the Gharb, the most malarious region of Morocco, are considerably lower than they were in 1932–34. An extension of irrigated areas in faulty conditions has in places extended endemic areas, but there has been an overall decrease of malaria intensity.

Norman White

BLAIR, D. M. **Report of Malaria Control in Southern Rhodesia.** *Pub. Health.* Johannesburg. 1951, May, v. 15, No. 5, 100–103.

Malaria and blackwater fever remain the most serious health problems in Southern Rhodesia though there has been some decrease in their incidence in Europeans during the last half century. Preliminary trials of residual insecticides gave promising results in 1946, and were followed by a major experimental scheme started in the Mazoe Valley in 1949. Teams of men undertook the control of both malaria and schistosomiasis, in summer and winter seasons respectively. The sole method used in malaria control was the distribution of insecticides, and the sole one in schistosomiasis control the destruction of snails by copper sulphate. Insecticides were sprayed on two occasions with a three-month interval ; on the first, DDT was used exclusively ; on the second BHC wettable powder (46 mgm. gamma isomer per sq. ft.) was substituted in African dwellings and in the surprisingly large proportion of European dwellings where it was accepted.

An assessment of the result on African health by morbidity and mortality statistics was not possible. The parasite rate at the end of the first year was 40·6 per cent. compared with 77·3 per cent. in adjacent untreated areas, and farmers reported a marked decrease in African sickness and a great improvement in the regularity of employees' attendance at work. The European hospital which had admitted an average of 78 malaria cases annually in the previous nine years, admitted 55 in 1949, and 2 in 1950, a nearby hospital showing no such decrease.

Two lines of attack are now being considered, both emphasizing local rather than central control. One includes the encouragement of local schemes carried out by Town and Village Management Boards, and the other a ten-year programme in the African reserves.

Figures of cost are given but slightly confuse the reviewer. The experimental scheme cost about three shillings and fourpence per hut or room per year. An estimate of 10d. per room per treatment is given for local authority work, and 1s. 8d. [per treatment or per year ?] for work in the reserves.

G. Macdonald

MASTBAUM, O. **Field Experiments with D.D.T. Emulsion and Wettable D.D.T. with special reference to Malaria Incidence in Swaziland during the Transmission Season 1949/50.** *East African Med. J.* 1951, Feb., v. 28, No. 2, 67–74, 1 graph.

The endemicity of malaria in Swaziland varies very greatly with altitude so that the country includes hyperendemic, epidemic, and intermediate areas. The vector is *Anopheles gambiae* and the predominant parasite *Plasmodium falciparum*. Small-scale control experiments in 1948–49 gave inconclusive results, and larger trials intended to rule out fallacies due to human migration were substituted in the following year. They covered 35,000 people, 2,500 square miles, and all types of endemicity. DDT was used in 3·5 per cent. emulsion, and as a suspension of wettable powder with a final DDT content of 5 per cent. The average dose was 0·43 gallons per hut, the average wall area of which is not stated. Parasite rates were taken in the non-malaria season before work started, and at monthly intervals throughout the season of control : the same procedure was adopted in untreated areas.

The average mosquito density was satisfactorily reduced in the treated areas. Parasite rates were consistently less in the treated areas than in the untreated ones. In those areas not classed as hyperendemic the rates in the malaria season remained at about their pre-season level without the expected

rise which occurred in the untreated places. In the hyperendemic areas there was a notable rise, though figures never reached the level attained where no treatment was given.

[Unfortunately the parasite rates for the non-transmission season, and for untreated areas, are not sorted out into hyperendemic and other zones as are those for the areas under treatment. It is therefore impossible for the reader to understand the amount of increase which occurred in some places, and to assess its significance.] *G. Macdonald*

WILKINSON, P. R. **Distribution and Fate of *Anopheles gambiae* and *A. funestus* in Two Different Types of Huts treated with DDT and BHC in Uganda.** *Bull. Entom. Res.* 1951, July, v. 42, Pt. 1, 45–54.

In 1949, Muirhead THOMSON [this *Bulletin*, 1949, v. 46, 439] cast doubt on the efficacy of DDT residual treatments for controlling *A. gambiae*, by demonstrating that these moquitoes could be caught alive in window traps in specially treated huts. This interesting experimental method has been used by other workers, and somewhat improved. Precautions need to be taken to prevent ants and other predators from removing dead mosquitoes and thereby falsifying the percentage kill observed. Special one-way entry " traps " are more satisfactory than the adventitious crevices on which Thomson relied, for they prevent accidental escape of live mosquitoes. The present author has repeated, in Uganda, the experiments against *A. gambiae* and *A. melas*, using mud and thatch huts of the Thomson type and also wood and fibre-board huts improved as described above. (Entry of predators was prevented by standing the huts on aluminium legs, resting on oil-soaked bricks.) The improved huts were sprayed with DDT and BHC dispersible powders to give deposits 3·3 and 1·5 times respectively those reported by Thomson. The mud huts were treated with 1·4 times the DDT dosage and 1·1 times the BHC dosage. The residual deposits were estimated chemically from test papers attached to the surfaces during spraying. The effects of these deposits were investigated by catches of live and dead *A. gambiae* and *A. melas* for 12 weeks before and 16 weeks after treatment. Every night a catcher slept in each treated hut, thereby acting as a human bait. Live and dead mosquitoes were collected in the morning from the floor sheet, the hut and the exit trap. The following is an overall summary of the percentages killed by the treatments :—

Treatment	Dose mgm. per sq. ft.	Percentage kill		Type of hut
		gambiae	*funestus*	
DDT	334	97	100	Wood and fibre board
gamma BHC	36	97	97	Wood and fibre board
DDT	193	62	78	Mud and thatch
gamma BHC	28	98	93	Mud and thatch

In the gamma BHC tests in the wooden huts, the few surviving mosquitoes were found inside the hut (they had probably entered shortly before dawn). In all other tests, the survivors were found in the exit traps. The total numbers of mosquitoes caught in the sprayed huts showed a distinct decline for several weeks after treatment, particularly in the BHC hut. This suggests a repellent effect which deterred some of the mosquitoes from entering.

While the BHC treatment appears to be superior at these doses, the difference does not appear to be so pronounced as reported by Thomson, who obtained a

complete kill with gamma BHC and only 2 per cent. with DDT. In an appendix, K. S. HOCKING points out that the failure of DDT in Thomson's trials seems to have been due to his using an unsatisfactory preparation of the insecticide, with large particles. Owing to sedimentation (both before and during application) the actual dosage on the walls would probably be much less than he estimated ; and no chemical checks were made. Furthermore, the large particles would be much less efficient in contaminating the mosquitoes. *J. R. Busvine*

SRIVASTAVA, R. S. **Malaria Control Measures in the Tarai Area under the Tarai Colonization Scheme, Kichha, District Naini Tal : September 1947 to December 1948. First Report.** *Indian J. Malariology.* 1950, June, v. 4, No. 2, 151–65, 1 folding map.

This preliminary report describes the malaria control measures that have been undertaken to render possible the development of a colonization scheme in the Tarai. The Tarai, a strip of land in the Naini Tal District of the United Provinces between the foothills of the Himalayas and the cultivated plain of Rohilkhand on the south, is a tract of forest and swamp with scattered patches of cultivated land. It has long had a most unenviable reputation for hyper-endemic malaria : malaria has indeed been the chief obstacle to the development of what otherwise might have been highly fertile country. The present development scheme concerns a block of territory some 30,000 acres in extent along the west side of the railway and road from Bareilly to Kathgodam and Naini Tal. There are 103 villages in this area of which 40 have been taken up for development as a preliminary measure. Only 23 of these 40 villages are inhabited ; their combined populations number 2,556. Spleen rates of children in these villages varied from 50 to 100 per cent. *P. vivax* is the predominant parasite. The percentage distribution of anopheline species among adults captured in catching stations was : *A. culicifacies* 56·2, *A. annularis* 15·8, *A. fluviatilis* 14·5, *A. splendidus* 7·4, *A. subpictus* 4·9 and *A. minimus* 0·5. The relative scarcity of *A. minimus*, a known vector of malaria in the Tarai, is noteworthy.

The control measures so far used have been DDT residual spraying and the mass distribution of paludrine [proguanil] tablets, 300 mgm. once a week for adults. The results have been promising ; dispensary attendances for malaria have been much fewer than in previous years. *Norman White*

RAO, T. R. **Malaria Control using Indoor Residual Sprays in the Eastern Province of Afghanistan.** *Bull. World Health Organization.* Geneva. 1951, v. 3, No. 4, 639–61.

The observations recorded were carried out in the Laghman District of the Eastern Province of Afghanistan from July to October 1949. The Laghman District is mountainous with several peaks rising to over 10,000 feet. The hills are rocky and bare but the valleys, some 2,000 feet above sea-level, are well irrigated. The two chief rivers of the district are the Alingar and the Alishing. They meet in the district and the combined river ultimately joins the Kabul River. The land adjoining the rivers is irrigated by canals which draw water direct from one or other of the two rivers ; the gradient of the country makes dams and reservoirs unnecessary. Melting snow on the high mountains provides a perennial flow in the rivers. Rounded boulders and pebbles compose the river beds : these ensure the formation of good anopheline breeding places whatever the water level may be. The rainfall is very scanty and most of it occurs in the winter. Very occasionally thunderstorms occur in July and August. Maximum temperatures range from 69·8°F. in midwinter

to 113°F. in midsummer, minimum temperatures from 29·8°F. to 69·8°F. The valleys are intensely cultivated, rice in summer, wheat in winter. Grapes and melons are grown in orchards in the upper valleys. From June to August, inclusive, irrigation is continuous ; it is intermittent in September and October. All houses are made of mud or sun-dried bricks : the flat roofs are made of logs overlaid with a thick coat of mud. Internal surfaces are very rough and very absorbent. A high rectangular mud wall encloses a courtyard into which all rooms open. Cattle often share the human living accommodation. From March to October excessive heat causes everyone to sleep outdoors. Water supply is abundant : a small water channel flows through the courtyard of almost every house. Sanitary conditions are primitive.

Malaria is hyperendemic throughout the area. In 14 unsprayed villages at the end of October the cumulative spleen rate was 89 per cent. (638 children aged 2–9 examined). In July–August, before spraying, the average parasite rate of 590 children in 15 villages was 18·8 per cent. (101 *P. vivax*, 10 *P. falciparum*, 1 *P. malariae* in 111 positive smears). In October parasites were found in the blood of 49 of 70 infants in unsprayed villages (70 per cent.).

The species of *Anopheles* found were : *annularis*, *culicifacies*, *fluviatilis*, *hyrcanus*, *maculatus*, *moghulensis*, *pulcherrimus*, *splendidus*, *stephensi*, *subpictus*, *superpictus*, *turkhudi* and *vagus*. *A. culicifacies* and *A. stephensi* were the most in evidence : they formed 42 and 34 per cent. of the 6,242 adult anophelines captured in random searches in unsprayed villages. *A. superpictus* formed 11 per cent. of the catch. Only *A. culicifacies* and *A. superpictus* were found naturally infected. None of the 252 *A. stephensi* dissected was infected ; it is a vector, however, in the neighbouring countries Baluchistan and Iran. The season of malaria transmission is believed to extend from May to October, being very intense in the last four of these months. Anopheline breeding was found in rivers, streams and swamps and in rice fields and channels during the irrigation season.

Thirteen villages, with a combined population of 14,000, situated between the Alishing and Alingar Rivers just above their junction, were sprayed in the first half of August. DDT-Aromex-soap emulsion and water wettable powders were used. Twelve weeks after spraying anopheline densities in sprayed villages remained under effective control. The average cumulative spleen rate in sprayed villages was 62 per cent. and in unsprayed villages 89·1. The infant parasite rate was 8·3 in sprayed villages as compared with 70 per cent. in unsprayed villages.

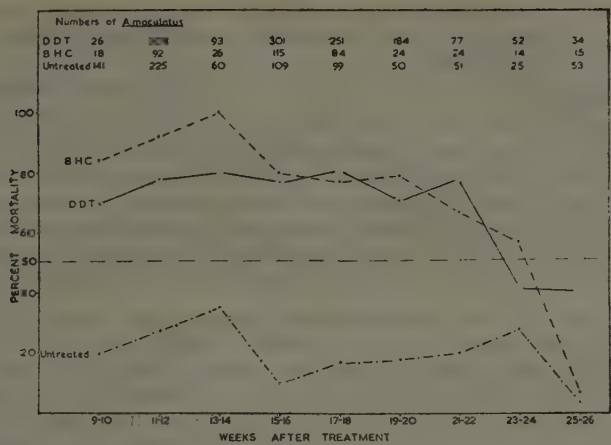
The results show that DDT spray is capable of controlling malaria in this part of Afghanistan. Though the inhabitants sleep outside at night throughout the transmission season the anopheline vectors appear to use human dwellings as their most important, if not sole, resting places. For complete control the first spraying should be done in May and a second in August.

Norman White

WHARTON, R. H. **DDT and BHC as Residual Insecticides in Malaya.** [Correspondence.] *Nature*. 1951, May 26, 854–5, 1 diagram.

WHARTON and REID previously reported experiments on the effect of DDT and Gammexane against *Anopheles maculatus* in Malaya, recording their results as a mortality over 12 weeks without subdivision into smaller periods [this *Bulletin*, 1950, v. 47, 321]. The experiments were repeated in 1950 with modifications in recording to show results at intervals of two weeks. They are illustrated in the graph below.

Culex fatigans was hardly affected by DDT, though BHC killed over 50 per cent. for 5 to 7 weeks after application.



48-hr. mortality of *A. maculatus* entering treated and untreated experimental huts. DDT, 200 mgm./sq. ft.; BHC, 40 mgm. gamma isomer/sq. ft.

[Reproduced from *Nature*.]

A. vagus and *A. aconitus*, which do not usually bite man, are common house-haunters in Malaya. They disappear from houses for about 6 months after application of DDT, but reappear in considerable numbers as early as one month after BHC. Possible explanations have been examined. It is shown that adults of these species captured from huts treated with BHC have a materially higher death rate in the next 24 and 48 hours than those from unsprayed houses, and this continues for 8 to 16 weeks after application of the insecticide. It seems that persistence in houses is not related to insecticide activity. *G. Macdonald*

WHARTON, R. H. **The Behaviour and Mortality of** *Anopheles maculatus* **and** *Culex fatigans* **in Experimental Huts treated with DDT and BHC.** *Bull. Entom. Res.* 1951, July, v. 42, Pt. 1, 1–20, 6 text figs. & 3 figs. on 1 pl. [18 refs.]

These experiments were undertaken to assess the value of DDT and BHC as residual spray treatments against *Anopheles maculatus* in Malaya. The experimental huts were fitted with window traps, as in the original type designed by Muirhead THOMSON [this *Bulletin*, 1949, v. 46, 439]. They were made of wood and palm thatch, lined with brown paper, and about 8×7×5 feet in size. To encourage entry of the not-too-plentiful *A. maculatus*, simple louvres were constructed to admit them easily, while excluding light. The huts were sited at the periphery of labourers' lines on an estate where *A. maculatus* was breeding uncontrolled. (The men were protected by suppressive proguanil.) Before the experiments, and throughout their progress, the mosquito population was sampled by regular trapping with a human bait.

Preliminary observations, by collectors in untreated huts, revealed the following points:

(i) Of 417 *A. maculatus* taken when feeding, 11 per cent. fed between 6 and 9 p.m.; 54 per cent. between 9 and midnight; 19 per cent. from midnight to 3 a.m.; and 16 per cent. between 3 and 6 a.m.

(ii) Observations of the window trap showed that of the total attempting to escape throughout the night, 19 per cent. were leaving by midnight and about 50 per cent. before dawn (6 a.m.).

(iii) Considerable numbers of mosquitoes were caught escaping through the entry louvres ; this showed that attraction to light was not the only factor influencing escape from huts.

Two trials with insecticides were done, the first in January 1948 and the second in March 1949. In the first trial, one hut was treated with DDT at 100 mgm./sq. ft. (from 33 per cent. wettable powder) and one with BHC at 20 mgm./sq. ft. (from 6·5 per cent. wettable powder). Two factors rendered the results of this trial very doubtful : (i) Large numbers of dead and dying mosquitoes were eaten by spiders and other predators, thus obscuring the numbers killed [this is an important matter, sometimes overlooked in this type of experiment]. (ii) The *A. maculatus* population was rather low. However, tentative conclusions were that the DDT was failing in 5–8 weeks and the BHC in 2–3 weeks.

In the second trial, *A. maculatus* was more numerous and the depredations of the predators were prevented as far as possible. Both treatments were applied at double the original rates, to clean paper linings. The kills of *A. maculatus* were assessed according to immediate kill and 24-hour kill of mosquitoes in the window traps. The DDT gave about 60 per cent. initial and 80 per cent. final kill, throughout a period of 16 weeks. The gamma BHC gave 100 per cent. kill for 4 weeks and then initial mortality fell to 19 per cent. in weeks 12–16, the 24-hour kill being almost complete up to 10 weeks and still 88 per per cent. at the 12–16 week period.

The DDT apparently had little effect on the feeding of *A. maculatus* ; but with the BHC there was a reduction in percentage fed.

DDT was relatively non-lethal to *Culex fatigans*, but had a marked irritant effect, driving them into the light trap. The biting rate was reduced from about 80 to about 45 per cent. BHC killed all *C. fatigans* which entered for 2 weeks, but had little effect on this mosquito by the 8th week. The biting rate was reduced to 27 per cent. for weeks 1–8 and most of the mosquitoes were found in the light trap.

There was some evidence that the BHC repels mosquitoes from entering the huts for one or two weeks after application.

In general, the results prove that *A. maculatus* is susceptible to deposits of DDT and gamma BHC, but in every respect, except persistence, gamma BHC at 40 mgm./sq. ft. was superior to DDT at five times that dose.

J. R. Busvine

J. National Malaria Soc. 1951, June, v. 10, No. 2, 97–196, numerous figs. [Numerous refs.] **Nation-Wide Malaria Eradication Projects in the Americas.** [Symposium held at 33rd Annual Meeting of the National Malaria Society, Savannah, Georgia, 8 November 1950.]

The National Malaria Society convened this symposium with the intention of emphasizing that it is now financially possible for a malarious nation to eliminate its malaria within a reasonable time. In so far as the Americas are concerned it has certainly achieved that objective, and has also emphasized that, except where bromeliad malaria is concerned, larval control in the Americas is as dead as the Dodo. The whole symposium should be studied closely by all directly concerned in major control schemes.

The survey covers four major countries, in each of which an effort is being made to reduce malaria in virtually all localities to the point of elimination. It is valuable both for the principles and the details involved. The schemes all justify the term "nation wide" ; the U.S.A. scheme covers all counties with a 5 per 100,000 malaria death rate, actually 332 counties in 13 States ; the Venezuela scheme covered in its last year 70·3 per cent. of all houses ;

the British Guiana one covered over 90 per cent. of the population exposed to malaria, and the Brazilian one the majority (not stated in figures) of the 17,000,000 people exposed to malaria, 2,364,279 houses being treated. So far as data are given the costs seem to be within the countries' easy economic reach ; the U.S.A. costs are not analysed *per caput* ; in Venezuela they amount to 89 U.S. cents per head per year ; in British Guiana 20·5 U.S. cents, and in Brazil 21·4 U.S. cents, though this latter figure appears to have been attributed to the entire population and restriction to that of malarious areas might more than double it. The results are well stated in each case, and in terms of the general vital statistics for the three South American countries. The most interesting of these are from British Guiana because they include an incidental control. Malaria is normally hyperendemic in that country, but in the years 1940 to 1942 it underwent a natural remission owing to exceptional climatic conditions which prevented the breeding of the main vector. The figures therefore cover two malarious and two non-malarious periods, one of the latter being natural and one due to deliberate control, but with changes closely resembling each other. A contrast of two years only is given : the last "average" year, 1945, and the last year of DDT control, 1949, shows the general trend in this and the other two South American countries reported on.

British Guiana. Vital Statistics 1945 and 1949.

Year	Birth rate	Death rate	Crude vital index	Infant mortality rate	Maternal mortality rate
1945	36·2	17·8	204	101	11
1949	41·4	13·3	312	77	5

The incidence of malaria itself is shown in several ways, and in each case seems to be approaching vanishing point ; this is perhaps best illustrated by the statement that in Venezuela the discovery of an acute positive case which is otherwise unexplained often leads to the apprehension of a smuggler who makes illicit journeys to uncontrolled countries.

Previously each of the countries had an anti-larval programme, all of which have been abandoned except that bromeliad malaria is still controlled by forest clearance in Brazil. The programmes are the outcome of deliberate plans. In the U.S.A. both anopheline eradication and control by mass therapy were rejected as impossibilities, and the plan covered elimination of the disease by mosquito control (as distinct from eradication) combined with rational therapy to be followed by a maintenance scheme. The problems in the later part of the first stage and throughout the second stage lie chiefly in the recognition of cases, and in consequence much of the recent development has been in the form of improvement of notification, provision of training for technicians in microscopic diagnosis and the follow-up and verification of notified cases, as notifications at this stage of work tend to exaggerate the cases and lead to misdirection of work. In the South American countries there has been a much greater tendency to look on anopheline eradication as at least worthy of consideration, a natural result of the initial success in British Guiana, and there is therefore more concern in residual anopheline prevalence and its measurement than in the North. Indeed one of the most interesting and valuable parts of the symposium is Giglioli's analysis of the bionomics of *Anopheles darlingi*, the factors which control its prevalence and distribution, and the probabilities

of its re-invading territories from which it has been cleared. This might well have been presented separately to prevent risk of its submergence in details of insecticidal procedure.

Dr. Paul F. RUSSELL, who presided, briefly introduced the symposium, pointing out that *A. albimanus* had been deliberately eradicated from Barbados twenty years ago. He emphasized the financial practicability of national schemes.

Justin M. ANDREWS reviewed "The Eradication Program in the U.S.A." (pp. 99–123) and gave the history of work in the U.S.A. up to the initiation of the National Malaria Eradication Program in 1947, the course of which has been recorded from time to time in this *Bulletin* [1949, v. 46, 111 and 440 ; 1951, v. 48, 430]. The objects are stated above, elimination of the disease followed by prevention of recrudescence. Insecticidal control is by DDT emulsion, applied in homes and privies at 200 mgm. per sq. ft. at varying periods which tend towards a single annual treatment. Recently great efforts have been made to improve standards of clinical diagnosis. They include the establishment of three Malaria Observation Stations, the training of technicians, and an elaborate follow-up scheme mainly operated by public health nurses with the backing of practitioners and of the laboratories. It is intended to continue these services after the apparent disappearance of clinical cases. The importance of this phase of work is shown by the fact that of 586 reported cases appraised in 1950, only 26 persons were considered to have suffered from malaria.

The changes in malaria morbidity and mortality are well shown in a series of graphs, but the author is cautious in their interpretation. Malaria was declining when the programme started, changes in notification procedure have made comparison difficult, and the domestic use of insecticides is increasing rapidly. But the existence of an accelerated decline in incidence since the inception of the scheme is beyond doubt. Whatever the reason the end of the first stage seems in sight.

Arnoldo GABALDÓN reviewed the "Progress of the Malaria Campaign in Venezuela" (pp. 124–141), partly by reference to a previous work [this *Bulletin*, 1950, v. 47, 4]. The outstanding characteristic of this scheme is the unusually full epidemiological data on which it is based, some of the more important points being recapitulated. The Central Malaria Division includes a well-equipped chemical laboratory which has been invaluable in maintaining standards of material. Serial chemical assays of wall deposits show that irregularities of distribution due to the mechanism of spraying machines make this method of appraisal unreliable. DDT is applied, usually as a suspension, otherwise usually as a solution and rarely as an emulsion, on a cycle which has tended to change from 100 mgm. per sq. ft. every 3 months to 200 mgm. every 6 months. There is, however, evidence that this period is too long, positive blood films becoming more numerous in the fifth and sixth months after spraying.

Results are checked by routine methods and by an organization for the discovery of cases of the disease. They include a marked reduction in all malariometric indices, an increased relative proportion of *P. vivax*, and a marked preponderance of discovered malaria cases in people living in houses which have been left unsprayed. *A. darlingi* is very greatly reduced in numbers, both in adult and larval stages ; *A. albimanus* larvae are not decreased in numbers although adults are, while the larvae of other common anophelines remain unreduced in number. Vital statistics follow the pattern illustrated from British Guiana.

The author concludes with a review of the economic and social effects of rapid improvement of health, which necessitate a broad outlook on the general welfare of the population.

George GIGLIOLI reported on the "Eradication of *Anopheles darlingi* from the Inhabited Areas of British Guiana by DDT Residual Spraying" (pp. 142–161) recounting the work described in this *Bulletin*, 1949, v. 46, 183, and elaborating it by more recent figures and a review of the bionomics of *A. darlingi*. It is an indigenous mosquito, very closely associated with man. In the densely populated areas it is most numerous in the vicinity of settlements. It seems impossible to make a definite statement that it does not occur wild in the forest, but it is closely associated with lines of travel and habitual camping sites. When these are deserted its survival may depend on the taking of animal food, but it appears that its existence there is secondary to that of man. Notable instances of the invasion of hitherto healthy sites are recorded. It is very sensitive to climatic conditions, particularly dryness, and its larvae to the sodium chloride content and hydrogen-ion concentration of water. It may therefore travel slowly over years along habitual routes, but its progression is impeded by the occurrence of certain types of water and might be set back for years or even decades by an unfavourable season. On the basis of this study and reasoning the policy of control, now that eradication from the main populated areas has been achieved, includes the integration of existing natural barriers with strategic DDT spraying of houses along routes through which it would have to travel to re-enter the coastlands.

Mario PINOTTI in his account of "The Nation-wide Malaria Eradication Program in Brazil" (pp. 162–182), recorded previous anti-larval work in Brazil, which for economic and administrative reasons was greatly restricted in scope. The chief vector is *A. darlingi*, though *A. aquasalis*, *A. albitarsis*, *A. cruzii* and *A. bellator* all play a part. The anti-larval campaigns against the last of these has continued, but that against all other species has been abandoned in favour of a DDT campaign, with an emulsion stabilized by a locally made emulsifier, and applied at the rate of 150 to 200 mgm. per sq. ft. usually once a year before the beginning of a season which lasts 6 to 8 months. The disappearance of all species from houses is noted immediately after spraying, and in some areas an extinction of the breeding of *A. darlingi* comparable to that seen in British Guiana has been noted. This is not, however, universal, profuse breeding continuing in some places, and records have been made of this mosquito selecting outdoor shelters in preference to houses. Results as measured by malariometric indices are very encouraging, the infant parasite rate approaches zero in most treated areas, and improvements in mortality and morbidity comparable to those seen elsewhere are set out.

Fred L. SOPER, dealing with the "General Principles of the Eradication Programs in the Western Hemispheres" (pp. 183–194), reviewed the history of eradication of mosquito-borne diseases, the shades of respectability the term has assumed in the malariologist's eye, and the vacillations in meaning between eradication of disease and of the vector itself. He then unashamedly states that eradication of the disease is well under way in the countries described above, and in some of them local eradication of the vector has occurred or is in progress. Work in the Americas is not confined to those countries; Argentina and Ecuador have comparable schemes, while Costa Rica, El Salvador, Guatemala, Honduras, British Honduras, and Nicaragua have similar objectives. The work needs internal organization such as has been described in connexion with each, and also an external co-ordinating mechanism, such as can be supplied by Pan American Sanitary Bureau or WHO, and should be extended and continued to include control of all house-haunting arthropods.

Criteria of Malaria Eradication.

The National Malaria Society in 1949 appointed a Committee to formulate criteria for use in determining when malaria ceases to be an endemic disease,

and the final report of this Committee is reproduced with all papers presented at the Symposium (pp. 195–196). The criterion of cessation of malaria endemicity which is advised is the cessation of indigenous cases for three years, provided that adequate reporting, including notification of names and addresses of sufferers, is actively promoted, and adequate investigations are carried out. The meaning of the terms used is further defined in the report, together with recommendations for the appraisal of evidence. *G. Macdonald*

DOWNS, W. G. & BORDAS, E. **Control of *Anopheles pseudopunctipennis* in Mexico with DDT Residual Sprays applied in Buildings. Part V. Effectiveness of Residual Applications of DDT and Gammexane up to One Year after Application under Controlled Conditions.** *Amer. J. Hyg.* 1951, July, v. 54, No. 1, 150–56. [25 refs.]

An earlier paper by the authors describes the diurnal activity of *Anopheles pseudopunctipennis*, which may be summarized as follows : it rests in houses during the day, leaves at dusk, feeds mainly out of doors and re-enters houses at dawn. A series of experiments was planned to investigate the irritant and lethal effects of residual insecticides on this mosquito, having regard to results recorded with other species.

Three identical houses with adobe walls were used, after six weeks' preliminary observations had proved that all were equally frequented by the mosquitoes. One was sprayed with DDT suspension, to leave a residue of about 200 mgm. per sq. ft. ; another was sprayed with BHC suspension to leave a residue of about 20 mgm. of the *gamma* fraction per sq. ft. ; and the third house was a control. Records were made of mosquitoes, both live and dead, in different stages of the feeding-oviposition cycle, inside the houses (on floor sheets) and trying to escape (caught in a window trap). The following conclusions were drawn from the results :—

1. Total numbers of mosquitoes entering the sprayed houses were less than those caught in the control ; this suggests a repellent action operating over a distance.

2. Of those mosquitoes which entered, irritation caused 35 per cent. to escape from the DDT and 20 per cent. from the BHC. The higher percentage escaping from DDT may be due to the phototropism it causes (which assists escape) and which is less evident, or absent, with BHC.

3. Of those mosquitoes which flew to the exit trap, 49 per cent. died after DDT and 85 per cent. after BHC within 48 hours ; these data compare with 24 per cent. deaths in controls.

4. The DDT residue remained effective for a long time (as long as 29 months in one case). BHC also remained effective for up to a year, though its speed of kill declined noticeably towards the end.

5. Elsewhere it has been shown that DDT residual spraying effectively reduces malaria in Mexico [this *Bulletin*, 1951, v. 48, 333]. From this paper it would seem that BHC would be at least equally effective. *J. R. Busvine*

PINOTTI, M. **The Biological Basis for the Campaign against the Malaria Vectors of Brazil.** *Trans. Roy. Soc. Trop. Med. & Hyg.* 1951, June, v. 44, No. 6, 663–82, 5 maps & 6 charts. [11 refs.]

An account is given of the varied methods used by the National Malaria Service of Brazil, in their dealings with malaria carried by mosquitoes with different ecological characteristics.

Fifty species of *Anopheles* are known in Brazil, of which nine have been found naturally infected. Five of the nine species are of major importance,

namely, *Anopheles* (*Nyssorhynchus*) *darlingi*, *A.* (*N*) *aquasalis*, *A.* (*N*) *albitarsis*, *A.* (*Kerteszia*) *cruzii*, and *A.* (*K*) *bellator*. The first three species (subgenus *Nyssorhynchus*) are responsible for endemic malaria north of the 25°S. parallel of latitude, which includes most of Brazil, while the subgenus *Kerteszia* is the malaria vector in an area of some 40,000 square kilometres south of that latitude.

In the *Nyssorhynchus* zone, malaria transmission in the interior is almost all due to *A. darlingi*, but in the coastal region it is mostly due to *A. aquasalis*, but with either or both of the other two species also taking part. *A. aquasalis* is also found as the main vector of malaria in a region 250 kilometres from the coast, where the soil is unusually saline. In the north-east, *A. darlingi* has only been reported from near the estuary of the São Francisco river.

Anopheles darlingi is the principal malaria vector in Brazil, and transmits the disease in the valleys of the major rivers and near impounded reservoirs. It prefers to breed in large, deep shaded reservoirs of cool water, with patches of floating plants such as *Pistia* and *Eichornia*. Floating islands of these plants may assist the dispersion of *A. darlingi* during floods. This species is the most domestic anopheline in Brazil, and is very susceptible to infection with the malaria parasite. In the valleys of the Amazon and São Francisco rivers it transmits malaria perennially, but, in some regions of São Paulo and Paraná, it is associated with epidemics every 5, 6 or 7 years, the adult mosquitoes disappearing in between these times.

Anopheles aquasalis prefers to breed in water having between 2 and 10 gm. per litre salt content, exposed to direct sunlight. Adults are produced in enormous numbers and the importance of *A. aquasalis* as a vector depends more on this than on any great preference for human blood. In certain zones of the north-east it has become a domestic insect. During the breeding season, adults may be found resting in houses, usually near the floor.

The variety of *A. albitarsis* which has been found naturally infected is *A. albitarsis domesticus*. This subspecies shows a marked preference for human blood. Infected specimens have been found only near the coast, and the varieties of *A. albitarsis* found inland are probably of no epidemiological importance. In two limited zones in the States of Bahia and São Paulo, *A. albitarsis domesticus* is reported to be the only vector present. Larvae have been found in many different types of habitat.

The anophelines of the subgenus *Kerteszia* are of great interest in that they breed in water among the leaf bases of plants of the family Bromeliaceae. Only in one other part of the world, the island of Trinidad, is "bromeliad malaria" an important problem. Of the two species important in the south of Brazil, *A. cruzii* predominates in the heavily forested regions, while *A. bellator* prefers the open coastal zone, but the division is not sharp. While bromeliad malaria is, in Trinidad, a problem of small rural areas, in Brazil it covers a large area, and is urban as well as rural. The kerteszias of Brazil are very much more domestic than those of Trinidad, and, in the region of bromeliad malaria, 90 to 100 per cent. of adults caught in houses are kerteszias. Their period of maximum activity is from midnight until dawn ; and they prefer to rest, after feeding, on the ceiling or higher parts of the wall. The National Malaria Service, created in 1941, began its campaign with larval control by means of Paris green, oil and minor drainage. Subsequently, house spraying with DDT, and the free distribution of chloroquine throughout the country, have been found effective in reducing blood parasite rates and malaria morbidity, as is shown by the charts. The mosquitoes are still by no means eradicated and pilot projects on larval control are in progress on certain coastal islands. In the zone of bromeliad malaria, the city of Florianopolis has been

freed from malaria by the manual removal of more than 20,000,000 bromeliads. In more densely forested regions where such removal is impracticable, deforestation around certain towns has produced good results. In the rural areas, chemical means of destruction of bromeliads are being sought. Spraying of forests with DDT and Gammexane from helicopters reduced the numbers of *A. bellator* and *A. cruzii* for a period of twenty days only. House spraying with DDT has been carried out in the south of Brazil and reduces malaria transmission considerably.

The distribution of the five major vectors is shown on the maps, and results are given in 6 charts and 2 tables. *A. J. P. Goodchild*

DE LUCENA, D. T. Profilaxia da malária com DDT inseticida no vale de Baixo São Francisco. Resultados do 1.° ano de experiência. [**One Year's Experience of Malaria Prophylaxis with DDT in the Valley of the San Francisco River, Brazil.**] *Rev. Brasileira Malariologia.* Rio de Janeiro. 1950, Apr. 30, v. 2, No. 2, 186–208, 7 figs. & 2 maps on 6 pls. & 3 graphs. English summary.

BECKER, E. R., SCHWINK, T. M. & BRODINE, C. E. **The Distribution of Sparing and Protective Factors in Plasma Fractions of Ducks recovered from Lophurae Malaria.** *J. Infect. Dis.* 1951, July–Aug., v. 89, No. 1, 16–25, 1 fig.

In earlier experiments the authors have described what they term a "sparing phenomenon" in chicks inoculated intravenously with washed duck cells parasitized with *P. lophurae* 1½ hours after receiving equal volumes of physiological saline or plasma of ducks, which had recovered from *P. lophurae* infections, by the same route. It was found that the chicks which had received the plasma showed from the time of inoculation up to 3 days afterwards a higher mean percentage of parasitized erythrocytes than those which received physiological saline. The explanation advanced was that the injected plasma "spared" the foreign parasitized cells from defensive action by the chick host. This "sparing" action was operative before passive immunity ("protective effect") from the injected plasma was apparent. TRAGER & MCGHEE [this *Bulletin*, 1950, v. 47, 816] have previously described the protection (passive immunity) afforded by the englobulin fraction of plasma of egg-laying hens for chicks infected with the same parasite. For the purpose of studying the various fractions from the plasma of recovered ducks the pooled heparinized or citrated blood of a number of ducks, cooled to 2–4°C., was separated into 4 fractions by using various concentrations of ammonium sulphate, as described in detail. The resulting parasitaemia in inoculated chicks treated with physiological saline or left untreated was then compared with those in chicks which had received various injections in physiological saline of one or other of the four fractions from plasma, as well as with combinations of these fractions. It was found that the "sparing factor" in the immune duck plasma, of protein character, occurs largely in the material precipitated by ammonium sulphate between the concentrations 33·4 to 50 per cent. The factor in immune duck plasma that conferred passive immunity, also of protein nature, was largely precipitated by ammonium sulphate at concentrations ranging from 0 to 33·3 per cent. By the use of ammonium sulphate it is thus possible to separate the "sparing factor" and the substance in which passive immunity resides. *J. D. Fulton*

BECKER, E. R., SCHWINK, T. M. & PRATHER, R. M., Jr. **Hemagglutinin-inhibiting Property of Duck Plasma exhibited in Agglutination Reactions involving Duck Erythrocytes and Plasma of Chicks recovered from Lophurae Malaria.** *J. Infect. Dis.* 1951, July–Aug., v. 89, No. 1, 95–102, 1 fig. [13 refs.]

The "sparing phenomenon" referred to in the abstract on p. 1084 is, according to the authors, not due to haemolysis, non-specific protein effect, blocking of the RE system, or lengthening of the lag phase of reproduction in the new host. In seeking farther for an explanation of the phenomenon it seemed possible that the sparing effecting might be due to excessive clumping of the duck cells injected to the chick so that normal phagocytosis did not result. On the other hand no evidence of these large aggregates had been observed in the smears of chicken blood which exhibited the phenomenon. The agglutinating properties of chick and duck serum were investigated further *in vivo* and *in vitro*. By the latter methods an effect, regarded as the counterpart of that *in vivo*, was observed, namely inhibition of haemagglutinins for parasitized duck red cells by the immune serum of chicks after recovery from infection with *P. lophurae*. The source of the antigen in duck plasma giving rise to this inhibition has not been accurately determined. It is, however, non-adsorbable on infected or non-infected duck cells. Interference with the action of fixation of complement or anticomplementary action in the haemagglutinin-inhibiting phenomenon has been excluded. The authors conclude that the sparing phenomenon observed *in vivo* is not due to the formation of excess haemagglutinin with resulting formation of red cell clumps. *J. D. Fulton*

BISHOP, Ann & MCCONNACHIE, Elspeth W. **The Inhibition by *p*-Aminobenzoic Acid of the Development of Paludrine-resistance as produced by Sulphonamides in *Plasmodium gallinaceum*.** *Parasitology.* 1951, July, v. 41, Nos. 1/2, 105–9. [14 refs.]

The production of resistance, in *P. gallinaceum* maintained in chicks, to paludrine (proguanil) and sulphonamides has been previously described [this *Bulletin*, 1947, v. 44, 969, 970; 1948, v. 45, 48, 1066; 1949, v. 46, 119] and cross resistance between these drugs has been noted. It is not yet clear how production of resistance to paludrine by sulphonamides is effected. The mode of action of the two types of compound may be related, but whereas the action of sulphonamides is inhibited by *p*-aminobenzoic acid (PAB) that of proguanil is not. It was considered possible by the authors that sulphonamides exert some effect on the parasite other than that antagonized by PAB and that this effect might produce the resistance to paludrine. Chicks infected with *P. gallinaceum* were therefore treated for 6 weeks with sulphadiazine in doses causing maximum inhibition accompanied by doses of PAB sufficient to antagonize the action of the drug. These doses were 5 mgm. of sulphadiazine and 0·5 mgm. PAB per 20 gm. twice daily by mouth. Resistance to sulphadiazine or to paludrine, however, did not develop, although similar doses of sulphadiazine in the absence of PAB were known to produce resistance to paludrine in a much shorter time. *J. D. Fulton*

MCCONNACHIE, Elspeth W. **Latent Infections in Avian Malaria in relation to the Production of Drug-Resistance.** *Parasitology.* 1951, July, v. 41, Nos. 1/2, 110–16. [30 refs.]

The resistance produced in *P. gallinaceum* to a variety of drugs has been frequently reported in the last few years. Success was only obtained, however,

when acute infections of chickens were used [this *Bulletin*, 1949, v. 46, 118; 1950, v. 47, 525]. Since in malarious areas patients with latent infections are frequently treated with antimalarials, the relationship of drug resistance to latent infections and the tissue phase of the parasites has been investigated by giving prolonged treatment with sulphadiazine to chickens latently infected with *P. gallinaceum*, and with paludrine [proguanil] to canaries similarly infected with *P. relictum*. Although resistance to paludrine is readily produced by sulphadiazine in acute infections it was not detected for either of these drugs after treating latent infections of *P. gallinaceum* in chickens twice daily with 20 mgm. of sulphadiazine per 20 gm. weight for periods of 171, 178 and 190 days. Resistance of *P. relictum* to paludrine was similarly absent after treatment of the host with doses ranging from 0·05 mgm. to 2·0 mgm. per 20 gm. daily over a period of one year. The author believes that failure to produce resistant strains of malaria by prolonged treatment of latent infections is indicative of the fact that such strains arise by selection of resistant mutants.

J. D. Fulton

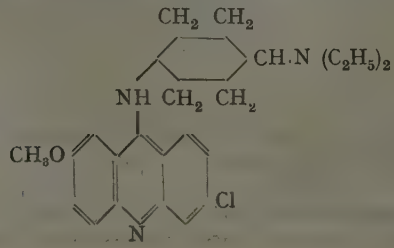
ROLLO, I. M. **A 2 : 4-Diamino Pyrimidine in the Treatment of Proguanil-resistant Laboratory Malarial Strains.** [Correspondence.] *Nature*. 1951, Aug. 25, 332–3.

The substance named in the title was one of a series of substituted 2–4-diaminopyrimidines whose activity in experimental malaria of laboratory hosts has recently been described by FALCO *et al.* [this *Bulletin*, 1951, v. 48, 872]. It has now been tested against a proguanil-resistant strain of *P. gallinaceum* and against two strains of *P. berghei* which had been made resistant to sulphadiazine and proguanil respectively. The former strain also showed a degree of resistance to proguanil as was noted in the case of *P. gallinaceum* similarly treated [this *Bulletin*, 1950, v. 47, 525]. It was found by the present author that the activity of the pyrimidine against the three resistant strains was practically unimpaired when compared with that against the parent strains. It also appeared that *P. gallinaceum* does not readily become resistant to the pyrimidine by the usual methods.

J. D. Fulton

ASANO, M., KAMEDA, Y., TAMEMASA, O., ISHII, N. & CHIDA, T. **Study of Antimalarials having Alicyclic Base as a Side-Chain. I. On 2-Chloro-5-(4′-Diethylaminocyclohexyl) Amino-7-Methoxyacridine.** *Japanese J. Exper. Med.* 1950, Dec., v. 20, No. 7, 779–88, 8 figs.

The chief difficulty in the synthesis of atebrin [mepacrine, quinacrine] and of plasmoquine [pamaquin] in good yield, is the introduction of the basic aliphatic side chain. The authors now describe the synthesis of a substance similar to atebrin, 2-chloro-5-(4′-diethylaminocyclohexyl)amino-7-methoxyacridine, having a basic alicyclic side chain, of the formula



Two cis-trans isomers were obtained. The hydrochloride of one believed to be the trans-form of orange-yellow platelets mp 292°C., the other of mp 288°C. consisted of yellow platelets, and was more soluble in water. Brief reports of the antimalarial activity were given by Ishii [this *Bulletin*, 1949, v. 46, 286] on *P. relictum* infections of canaries, using the Roehl technique, with similar or slightly better results than those given by atebrin. The trans-form appeared to have a slightly better therapeutic index. Seven cases of *P. vivax* infection were also treated with one or other of the above isomers, but the results are inadequately described and it is impossible to arrive at any conclusions from the data given. The authors, however, consider that the results were similar or slightly inferior to those obtained with atebrin. Unpleasant side effects were absent and the skin was not coloured. *J. D. Fulton*

Erratum

In the abstract relating to the Report of the Malaria Conference in Equatorial Africa published in this *Bulletin*, 1951, v. 48, 697–699, the word " permanent " on the third line of paragraph 4, page 699, should read " prominent ".

TRYPANOSOMIASIS

Roubaud, E. & Provost, A. Persistance des caractéristiques raciales d'une souche neurotrope de *Tr. gambiense* après passage en infection septicémique chez l'homme. [**Persistence of Racial Characteristics in a Neurotropic Strain of** *T. gambiense* **after Passage through Man.**] *Bull. Soc. Path. Exot.* 1951, v. 44, Nos. 3/4, 174–8, 2 figs. on 1 pl. [10 refs.]

In 1934 a strain of *Trypanosoma gambiense* was isolated from man in Yaoundé (French Cameroons), and has since been maintained in laboratory animals. This strain is characterized chiefly by its neurotropic character, manifested in the production in laboratory rodents of encephalomyelitis and paralysis. The course of infection is generally slow but it can be accelerated by intraperitoneal passages. Furthermore, the trypanosomes of this strain differ morphologically from those of other laboratory strains of *T. gambiense* in greater length (average 28μ). On account of the stability of all these characters it is regarded as a distinct subspecies.

An accidental infection of one of the authors with this strain provided the opportunity for studying its behaviour after passage through man. In the human host, trypanosomes were very numerous by the 13th day, producing parasitaemia, which was cleared by treatment with lomidine [this *Bulletin*, 1949, v. 46, 917]. Before treatment the trypanosomes were passaged through rats and guineapigs, in order to detect any changes in the strain after its passage through man. At the beginning, the infections ran a more rapid course, with numerous parasites, killing two rats in 57 and 73 days respectively. In subsequent passages through rats and mice the infections gradually reverted to their usual duration. During the first two years after their sojourn in man, there was not a single case of paralysis among the infected laboratory animals, but by the end of the third year two out of four mice manifested typical paralysis of the hind quarters. On the other hand, there was no change in the morphology of the trypanosomes, which remained longer than those in all the other laboratory strains of *T. gambiense*. It is concluded that, after passage through man, the Yaoundé strain did not lose any of the characteristics which

it has shown during fifteen years of maintenance in laboratory rodents. In view of the constancy of these characters, it is proposed to name this strain *T. gambiense* var. *yaoundé*.

With regard to the question whether this strain occurs under natural conditions, or was produced as the result of therapeutic treatment or artificial passages through rodents, the authors advance the following arguments: (1) 2 years after its original isolation from man it was still capable of developing in *Glossina palpalis*; (2) after 5 years it was still culturable in modified Razgha's medium; (3) from these observations it follows that in the early stages of its laboratory life, when this strain already manifested all its biological peculiarities, it showed evidence of close adaptation to its natural intermediate host. Hence it is concluded that this strain already possessed these characters under natural conditions.

C. A. Hoare

HAWKING, F. & THURSTON, June P. **The Persistence of Metacyclic Trypanosomes in the Blood.** *J. Trop. Med. & Hyg.* 1951, Sept., v. 54, No. 9, 185–6.

This experiment was incomplete owing to lack of infective tsetse flies; its aim was to see whether metacyclic trypanosomes, like malarial sporozoites, leave the blood during the incubation period of the disease or develop directly in the blood into the ordinary blood forms. Tsetse flies (*Glossina morsitans*) were fed on guineapigs infected with *Trypanosoma rhodesiense* and one fly only was found to be infective; its "intestines and salivary gland" were injected intravenously into two mice and, one hour later, the blood of one of the mice was inoculated into three other mice which all became infected. The authors concluded that the infection of these three subinoculated mice was caused by trypanosomes from the salivary glands of the fly, the trypanosomes having apparently remained in the blood of the mouse for one hour. [No reason is given for injecting the intestines of the fly; BROOM and BROWN (this *Bulletin*, 1937, v. 34, 930) infected a mouse by inoculating intestinal forms of a strain of *T. brucei* which was untransmissible by tsetse flies, the last feed of the fly on an infected animal having been taken 22 days before the fly was dissected. As they succeeded only once in 36 trials it is very unlikely that infection in the present experiment was caused by trypanosomes from the intestines of the fly.]

J. F. Corson

RAGEAU, J. Tsés-tsés et végétation au Cameroun français. [**Tsetse Flies and Vegetation in the French Cameroons.**] *Bull. Soc. Path. Exot.* 1951, v. 44, Nos. 5/6, 302–6, 8 figs. on 4 pls. [18 refs.]

The author notes that in the French Cameroons the riverine and lacustrine vegetation in the breeding places of *Glossina palpalis* is similar to that described for the Gold Coast by MORRIS [this *Bulletin*, 1947, v. 44, 51]. Since, however, a single species of tree or bush may vary considerably in its form according to the conditions under which it grows, more importance is attached by the present author to the general form of trees and bushes than to their botanical identity, in selecting the individuals to be cut down for tsetse control. Precise botanical study is, however, indicated in savannah country where it is desirable to recognize and cut out drought-resisting and fire-resistant species of trees and shrubs, which provide breeding places and shelters for fly in the dry season. There is a brief account of the vegetational characteristics of some localities where one or more species of *Glossina* have been found.

Glossina palpalis occurs in the dense shade and high humidities of secondary forest along the banks of streams. The flora includes large-leafed trees

(*Myrianthus arboreus*, *Mitragyna macrophylla*, *Anthocleista nobilis*, *Vernonia conferta*, and an unidentified croton) ; large-leafed or rambling bushes (*Neoboutonia velutina*, *Alchornea cordiflora*, *Rauwolfia* sp., *Conopharyngia*, *Voacanga*, etc.) ; young palms with basal fronds (*Elaeis guineensis*) ; creepers (*Paullinia*, *Cissus*, *Ampelocissus*, *Dioscorea*, *Ipomoea* and *Smilax*) ; herbaceous plants with tall stems (*Costus*, *Aframomum*, *Thalia* and *Pennisetum*). Some trees such as *Albizzia* and *Musanga* occur. There is little seasonal change in the vegetation. This species of tsetse fly is also abundant at a spring where there is dense vegetation comprising young *Musanga* trees, bushes, creepers, tree-like ferns (*Raphia vinifera*) and various tall herbaceous plants. *G. brevipalpis* occurs with *G. palpalis* in forest lining river courses passing through more open savannah country. Shade and high humidity are provided by the association of tall trees, and the dense undergrowth of bushes, creepers, and tall grasses.

G. palpalis fuscipes and *G. haningtoni* have been taken in a riverine forest consisting of widely spaced trees with branches about 40 metres above ground beneath which lies a dense vegetation of bushy shrubs, creepers, and smaller trees with branches about 4–5 metres from the ground. *G. caliginea*, together with a dark race of *G. palpalis palpalis* and *G. pallicera* occur in coastal mangrove swamps of *Avicennia*, *Rhizophora* and *Pandanus*.

G. tachinoides occurs in light fringing forest and savannah country in the north. Spiny bushes and bushy shrubs extend along the river courses and into the drier terrain behind the rivers. *Mimosa asperata* and *Sesbania punctata* are characteristic species, but the fly is not so closely associated with the dwarf willow (*Salix ledermannii*) or *Acacia* bushes. The fly is restricted to the riverine zone in the dry season but extends its range during the rains.

Eight photographs illustrate the various types of vegetational association described in the text. *D. S. Bertram*

CATES, J. E. & MCILROY, M. B. **African Trypanosomiasis in a British Soldier.** [Memoranda.] *Brit. Med. J.* 1951, Aug. 18, 401, 1 fig. on pl.

A soldier aged 30 fell ill in the Gambia in June 1942. Malaria was diagnosed. The same condition was again diagnosed a year later, and he was invalided home. In 1944 a swelling appeared at the side of the neck with fever, but this subsided in a few days without treatment. In May 1947 a tender swelling appeared in the right axilla and the spleen, and other lymph nodes were enlarged. He was admitted to hospital, being afebrile at the time.

A lymph node was removed from the side of the neck and sections showed a lymphoid follicular reticulosis. He was treated by X-rays to the neck, axilla, groin and spleen. In December 1947 he reported with fever and a rash, both of which recurred repeatedly, and in May 1948 he developed a pneumococcal septicaemia which responded to penicillin. An orchitis developed together with enlarged glands in the neck and an enlarged spleen. Trypanosomiasis was considered as a possible diagnosis, and the blood was repeatedly examined but found to be negative. In September 1948 there was lung consolidation and a recurrence of pneumococcal septicaemia. There were signs of meningitis with a grossly changed cerebrospinal fluid. The pneumococcal infection responded to penicillin.

A therapeutic trial with suramin was then made and the patient's condition improved. He was therefore given a full course of suramin treatment and remained well for a year, but was readmitted in May 1950 with intermittent fever and splenomegaly. Trypanosomes were at last found in the blood and in sub-inoculated guineapigs and mice. There was no clinical evidence of involvement of the nervous system, but the cell-count and protein-content of

the cerebrospinal fluid were increased. Suramin and tryparsamide were given and the patient has since remained well. *E. M. Lourie*

CHESTERMAN, C. C. ; MCGOWAN, D. ; ROBERTSON, K. **Diagnosis of Trypanosomiasis.** [Correspondence.] *Brit. Med. J.* 1951, Sept. 1, 546–7.

These letters were prompted by the report of a case of trypanosomiasis in a European soldier by CATES and MCILROY [p. 1089].

Chesterman points out that there is no record of the accepted method of diagnosis—gland puncture—having been practised at any time during the six years when the patient suffered from irregular pyrexia and cervical lymphadenitis. This technique should surely have made the diagnosis clear.

McGowan makes a similar comment, and gives a detailed description of the gland puncture technique as practised by field workers in West Africa [described also by HARDING & HAWKING, this *Bulletin*, 1945, v. 42, 188].

Robertson reports another case of a European, seen in England in March, 1948, five months after returning from West Africa, in whom enlargement of the lymph glands first suggested one of the reticuloses, so that a lymph node was excised for sectioning. Gland puncture later revealed living trypanosomes. The cerebrospinal fluid showed increases of protein-content and cell-count. Suramin was given and the patient is believed to have recovered. [This would be exceptional because it is well established that suramin alone is normally inadequate for the treatment of sleeping-sickness which has already advanced to the stage where there are changes in the cerebrospinal fluid.]

E. M. Lourie

KLEINSCHMIDT, A. Über den Feinbau von Trypanosomen. [**The Finer Structure of Trypanosomes.**] *Ztschr. f. Tropenmed. u. Parasit.* Stuttgart. 1951, Apr., v. 2, No. 4, 507–12, 5 figs.

The author reports the results of further observations on the cytology of trypanosomes. The methods used in the previous study [this *Bulletin*, 1951, v. 48, 622] have been superseded by osmium-fixation of the preparations used for observations by light and electron microscopy.

In the present study *Trypanosoma lewisi* was used for observations on the inner structures, and *T. brucei* served for the examination of the surface structures. The trypanosome-infected rat or mouse blood was mixed with Ringer's solution containing 0·5 per cent. glucose, after which fibrin was removed and the suspension centrifuged. For observations of the cytolytic action of hypotonic solutions, 1 cc. amounts of the suspension were placed in watch glasses with increasing quantities of distilled water. The selected dilution of the suspension was then fixed with osmic acid and washed in distilled water. In the case of *T. lewisi*, small samples were spread on very thin collodion films, which were examined with or without previous metal shadowing. In the case of *T. brucei*, the best results were obtained with metal shadowed smears spread on coverslips. In the course of these manipulations the flagellates disintegrate in various degrees—according to the methods used—revealing the finer structure of their body.

These observations have shown that the body of the trypanosomes is invested in a membrane or periplast consisting of fibrils, the arrangement of which in *T. lewisi* is denser than in *T. brucei*. By light-microscopic methods this periplast can be faintly detected after staining, especially in crushed preparations and after treatment with gallic acid. The surface of the body then appears to be basophile, which accounts for the fact that the trypanosomes retain their motility only in neutral and alkaline media. Owing to the non-transparency

of the body when viewed with the electron microscope, the structure of the nucleus and kinetoplast cannot be made out clearly. As regards the difference in the fibrillar arrangement on the surface of the body, observed in the two trypanosomes, it accounts for the difference in their mode of locomotion : in the larger frog-trypanosome these structures are correspondingly coarser. The examination of disintegrated fibrils shows that they consist of minute (60 mμ) hexagonal platelets arranged in the form of a string of beads, while in damaged trypanosomes the flagellum also reveals a fibrillar structure.

C. A. Hoare

POLSON, A. **Effect of Diseases on the Serum Albumin of Animals.** [Correspondence.] *Nature.* 1951, Sept. 8, 421–2, 2 figs.

GIARMAN, N. J. **Antibiotic Lactones and Synthetic Analogs. III. Efficacy in Experimental Trypanosomiasis.** *J. Pharm. & Exper. Therap.* 1951, July, v. 102, No. 3, 185–95, 1 fig. [18 refs.]

A review of the literature reveals that lactones derived from plant or animal sources possess a wide range of physiological activity. Little appears to have been done in estimating their activity against protozoa. A number of well-known substances possessing a lactone structure, and penicillin with lactam structure, have been shown to be without action on protozoa. This investigation was carried out to find how variations in the lactone structure, including the position of unsaturated bonds and the presence of various substituents, affected activity. Twelve furanes with formal structure superficially similar were also examined for activity in trypanosome infections. For this purpose albino mice of same age and sex were infected intraperitoneally with 10^6 parasites (*T. equiperdum*) and treatment was begun in a few hours except when the effect of delayed treatment was being studied. The drug in doses of $\frac{1}{4}$ to $\frac{1}{2}$ LD50 were administered intraperitoneally or subcutaneously. The observation period for estimation of survival rates was 21 days, since death in control untreated animals occurred within 48 to 120 hours. An increase in survival time was used as evidence of drug activity. Twenty-four lactones were tested but no cures resulted, although death of the mouse host was delayed by 5 of these substances. Some conclusions were drawn. Among the 12 furanes tested, some substances effected a limited number of cures including furfuryl alcohol, furfuraldehyde and furoic acid. Activity was abolished by saturation of the molecule through reduction. The introduction of a nitro-group in position 5 of the furane nucleus such as occurs in the semicarbazone of 5-nitrofurfuraldehyde (Furacin), previously reported in the literature, led to increased activity.

J. D. Fulton

MONTALVÁN C., J. A. Algunas consideraciones sobre el problema de la enfermedad de Chagas en el Ecuador. [**The Problem of Chagas's Disease in Ecuador.**] *Rev. Ecuatoriana de Hig. y Med. Trop.* Guayaquil. 1950, Jan.–June, v. 7, Nos. 1/2, 1–6, 1 map. English version 7–12.

In 1929 ARTEAGA reported cases of Chagas's disease from St. Elena and the neighbourhood, but since then the only place where the disease was thought to exist in Ecuador was Guayaquil. Then, in 1949, LEÓN recorded cases in the Province of Manabí, in the littoral and Andean regions. The author then started to examine persons from different parts of the country to find out whether other foci of the disease existed and found them in the Provinces of Guayas, Manabí, Los Rios, El Oro, Loja, Azuay and Bolívar. In a table are

given the numbers examined and the numbers giving a positive complement-fixation reaction. In Guayas Province, at General Vernaza, 3 were positive of 96 samples, and at Salitre 22 out of 194 ; in Manabí Province, of 9 localities examined 5 were positive, and 15 out of 247 samples ; 5 out of 24 in Manta, 6 out of 102 in Chone, 2 out of 52 in Portoviejo, and 2 out of 18 in Jipijapa. In Los Rios, one table gives three places where positive tests were obtained, namely Quevedo, Babahoyo and Palenque, but in another table, of 28 samples examined from this Province none was positive ; in El Oro, at Zaruma 9 were positive out of 31 ; at Machala 4 out of 30 and at Pasaje 2 out of 5 ; in Loja, 2 out of 15 at Trapichillo and 2 out of 30 at Valle Hermoso. The author's aim was, however, not to discover the degree of prevalence, but merely the presence of the disease.

Six species of *Triatoma* have been identified in Ecuador : *T. dimidiata* (the only one found naturally infected), *T. carrioni*, *T. coxorufa*, *T. rubrotuberculatus*, *T. venosa* and *Rhodnius prolixus*. DDT has proved very effective in reducing the numbers of *Triatoma* in Guayaquil. This is of particular interest because others have reported that DDT is ineffective against *Triatoma* and may even prove harmful by getting rid of certain insects which destroy *Triatoma* eggs and so favour the multiplication of the *Triatoma* itself. *H. Harold Scott*

SENECA, H. & HENDERSON, E. **Growth of Hemoflagellates in Dialyzing Sacs and the Production of Growth Inhibitory Factor.** *Amer. J. Hyg.* 1951, Jan., v. 53, No. 1, 17–21, 2 figs.

Dialysing sacs have previously been successfully employed for the cultivation of *Trypanosoma cruzi* and for certain bacteria, in the latter case resulting in a high yield of toxin. With similar ends in view, the authors have undertaken a study of *T. cruzi* and *Leishmania donovani*. The apparatus used for their cultivation consisted of a dialysing chamber, from the upper end of which was suspended a bag made of seamless cellulose sausage tubing. Its lower end was tied in a knot or invaginated upwards and tied outside the chamber. Glass beads were put into the sac for stirring its contents. Both the chamber and sac were filled up to a certain level with a fluid culture medium made up as follows : 50 gm. Bacto-beef is mixed with 1 litre of water for one hour at 50°C. and 5 minutes at 80°C. and then cooled ; it is then filtered, and 20 gm. of neopeptone + 5 gm. NaCl are added, *p*H is adjusted (7·2–7·4) and the volume is restored to 1 litre. After autoclaving, this mixture serves as stock medium, to 9 parts of which 1 part of rabbit serum is added before use.

Cultures of the trypanosome and leishmania are initiated by inoculating the medium contained in the sac, with addition of 50,000 units of penicillin to prevent contamination. Control cultures in the same medium were maintained in a flask. All cultures were kept at room temperature, and the medium in the sac was changed once or twice daily. For counting the flagellates, samples of about 0·5 ml. were removed from the culture, transferred to a blood-counting chamber, and heated gently (50–60°C.) to kill and fix the flagellates. The counting was then carried out as in the case of blood cells, while the appearance of the flagellates was studied in fixed smears.

For the study of the inhibitory factors produced by the haemoflagellates, the medium in the chamber was not renewed but at weekly intervals for 8 weeks a portion was removed, and inoculated with cultures of the trypanosome and leishmania.

Growth of the haemoflagellates in the dialysing sac was considerably more abundant and of longer duration than in the control flask. As regards the effect of the dialysate, it was found that the growth of each of these haemoflagellates was inhibited by the dialysate from cultures of its own species, but

not from those of the other, though sometimes dialysates from old cultures had a slight inhibitory action on the heterologous organisms. The authors conclude " that the inhibitory principle is dialyzable, type specific, and in some probability a product of autolysis of the organism ". *C. A. Hoare*

WOOD, S. F. **Importance of Feeding and Defecation Times of Insect Vectors in Transmission of Chagas' Disease.** *J. Econom. Entom.* 1951, Feb., v. 44, No. 1, 52–4.

The successful transmission of Chagas's disease (*Trypanosoma cruzi*) depends on the contamination of wounds, abrasions, or mucous membranes, of the host, with infective faeces of blood-sucking Triatomid bugs. Transmission may also occur when bugs are crushed on the skin.

The efficiency of any particular species of bug as a vector of Chagas's disease will, therefore, be affected by, on the one hand, the rapidity with which defaecation follows feeding, and on the other, by the length of time which the bug spends in feeding, and exposed to the risk of being crushed by movements of the host.

Four species found in California, namely *Triatoma protracta*, *T. rubida uhleri*, *T. longipes*, and *Paratriatoma hirsuta*, have been compared from this point of view.

Some records of the length of feed at different temperatures are given in the text. The range is from 2 minutes 50 seconds at 93°F. for *P. hirsuta* to 29 minutes 10 seconds at 83°F. for *T. protracta*. No mention is made as to whether a comparable degree of engorgement was reached by each bug timed. The records concerning defaecation are much more extensive, and are given in the form of a table.

Twenty-two bugs were observed, and the time, after feeding, at which defaecation occurred, was recorded for up to eight successive defaecations from each bug. The temperature was also recorded, and on two of the bugs (*T. protracta*) observations were made at two different temperatures. In four of the experiments with *T. protracta*, no defaecation occurred during the period under observation, the times at which observation ceased being 223 and 221 minutes at 66°F., 180 minutes at 91°F. and 89 minutes at 94°F. These figures are not included in the tabulated data.

From the table, it is apparent that *T. rubida uhleri* commences defaecation sooner, and defaecates more frequently, than the other three species, the time from feeding to first defaecation averaging 1 minute 37 seconds at 91°F., while *T. protracta* averages 6 minutes 13 seconds at 94°F. and the other two species range from 25 to 63 minutes at these temperatures.

It is concluded that *T. rubida uhleri* is the most likely species to transmit Chagas's disease through its faeces, while *T. protracta* is most likely to be crushed by the host and to transmit the disease in that manner. *A. J. P. Goodchild*

RODRIGUEZ M., J. D. Inmunidad en la enfermedad de Chagas. La reacción de fijación del complemento. [**Immunity in Chagas's Disease. Complement-Fixation Reaction.**] *Rev. Ecuatoriana de Hig. y Med. Trop.* Guayaquil. 1950, Jan.–June, v. 7, Nos. 1/2, 65–74. English version 75–83.

The author discusses the different methods of diagnosing Chagas's disease, namely by direct examination of the blood fresh or in stained smears, inoculation of blood into susceptible animals, cultural methods, and xenodiagnosis, and shows that none of these is altogether satisfactory, giving examples of the proportions in which these means fail. He then proceeds to the consideration

of the complement-fixation test, speaking in turn of the Machado-Guerreiro antigen of trypanosomes from infected animals, of *T. equiperdum* used by Muñiz and Freitas, of Kelser's culture emulsion in Bonacci's medium in glycerin, of Romaña and Diaz's alcoholic culture extracts, of Davis's watery suspensions of cultures, preserved with merthiolate after alternate freezing and thawing, and of Senekjie's flagellar and somatic antigens.

The author's tests have been made with the penultimate of these, Davis's method, later transplants into Muñiz' medium after preliminary culture in N.N.N. medium.

Samples of blood were taken from persons attending various departments of the Guayaquil city hospital and sera sent by practitioners from suspected cases of Chagas's disease. Altogether 606 specimens were examined: 53 from outside physicians, of patients who gave a history of palpebral oedema, recent or in the past and so were suspicious cases; 384 hospital patients with no obvious indications of infection by trypanosomes; 37 samples sent to the National Institute of Hygiene; 28 from patients under treatment at another hospital; 72 " sent by other physicians " and 32 apparently healthy subjects. Excluding the 53 first-named, there were 46 positive among the remaining 553 (8·3 per cent.). There was none among the healthy controls or in the 28 from the Vinces hospital in the Province of Los Rios; 3 among the 37 specimens from the Institute of Hygiene; 16 among the 72 specimens " from other physicians " and 27 from the 384 hospital patients.

On the 53 with past or present oedema of the eyelids one or more of four examinations were carried out: direct examination, inoculation, xenodiagnosis and complement fixation, and the results of each are presented in a table. Fifty of them were examined by the direct method, 18 were positive; 29 by inoculation, 14 were positive; 20 by xenodiagnosis, 3 were positive; 48 by complement fixation, 17 were positive. An interesting point is that two patients with *mal de engasgo* both reacted positively. This condition and its possible connexion with infection by *T. cruzi* have been noted on previous occasions.

H. Harold Scott.

MANRIQUE I., J. Aspecto clínico local de la cardiopatía chagásica crónica. [**Local Clinical Aspects of Cardiopathy in Chronic Chagas's Disease.**] *Rev. Ecuatoriana de Hig. y Med. Trop.* Guayaquil. 1950, Jan.–June, v. 7, Nos. 1/2, 13–39, 18 figs. English version 40–64.

BACKHOUSE, T. C. & BOLLIGER, A. **Transmission of Chagas' Disease to the Australian Marsupial** *Trichosurus vulpecula.* *Trans. Roy. Soc. Trop. Med. & Hyg.* 1951, Apr., v. 44, No. 5, 521–33, 6 figs. on 3 pls. [10 refs.]

The authors record the results of experimental infection of 40 specimens of the Australian possum or phalanger, *Trichosurus vulpecula*, with *Trypanosoma cruzi*, a culture of which was first inoculated into one of the marsupials, from which blood-passages—by the intraperitoneal, intramuscular, or subcutaneous routes—were made through a series of these animals and one echidna, *Tachyglossus aculeatus.*

The infection succeeded in all the animals used, producing an acute form of Chagas's disease, characterized by fever (upwards of 100°F.), tachycardia, dyspnoea and a high mortality rate, 60 per cent. dying in 21 to 35 days after inoculation. Those animals which survived for 35 days continued to live indefinitely.

Trypanosomes usually appeared in the blood on the 11th day, disappearing from the circulation 11 days later. The animals might die without any

parasites in the blood, but sometimes the blood would be teeming with trypanosomes. In recovered opossums the blood stream was free of parasites, and no infection resulted from the inoculation of their blood into normal animals.

From post-mortem examination it was evident that the cause of death in the experimental animals was heart failure. A histopathological study, based on 23 animals, was made of sections of the heart, spleen, liver and kidney, and some other organs. Masses of developing parasites in the leishmanial stage were found in the heart muscle, and smaller groups were occasionally seen in the skeletal muscles. Very small numbers were encountered in the spleen and liver, but none in other organs. In infected animals dying on or after the 35th day, parasites were usually either scanty or absent in the hearts, but these showed evidence of myocardial lesions.

Re-inoculation of *T. cruzi* in 6 opossums, which had recovered from Chagas's disease, failed to infect them, thus producing evidence of acquired immunity. However, attempts to produce passive immunization, by inoculation of 12 opossums with the blood of recovered animals, were unsuccessful, for they all proved to be susceptible to infection with the trypanosome, though their mortality rate was reduced.

The authors regard the Australian phalanger as a suitable animal for the study of immunity.

The changes in the heart and myocardium are shown in six photographic illustrations. *C. A. Hoare*

AVILÉS NUGUÉ, F. & RÍOS, T. Infección de la rata blanca a través de la mucosa oral con Tripanosoma cruzi. Reporte preliminar. [**Infection of White Rats with *Trypanosoma cruzi* through the Oral Mucosa. Preliminary Note.**] *Rev. Ecuatoriana de Hig. y Med. Trop.* Guayaquil. 1950, Jan.–June, v. 7, Nos. 1/2, 84–90, 4 figs. English summary.

GOBLE, F. C. **Studies on Experimental Chagas' Disease in Mice in relation to Chemotherapeutic Testing.** *J. Parasitology.* 1951, Aug., v. 37, No. 4, 408–14, 1 fig. [21 refs.]

In this investigation the author has made observations on the course of *T. cruzi* infections in 3-weeks-old mice inoculated with parasites grown in culture, instead of by the more usual method of using infected blood, in chemotherapeutic tests. Two strains of *T. cruzi* were used, named respectively A and B. The medium described by JOHNSON [*J. Parasit.*, 1947, v. 33, 85] or slight modifications thereof were used to maintain cultures. The infective material was generally obtained from the culture overlay after 2 weeks' growth and 30 million organisms were inoculated intraperitoneally in chemotherapeutic tests, since it appeared that smaller numbers gave less suitable infections. Under these conditions death of the host occurred sometimes within 10 days.

Drug administration by the intraperitoneal route was begun four days after infection and continued once daily for 6 days, before early deaths occurred in the mice, at a dosage level equivalent to the intravenous LD50, which was generally well tolerated, but modification of these conditions was sometimes necessary. The survival rate at the end of a period of 60 days was used as an indication of drug activity. The virulence of strain A for mice was unimpaired after 5 months in culture and caused 100 per cent. mortality. After one year in culture only a 20 per cent. mortality resulted and the survival period was increased. Virulence was subsequently enhanced by prolonged passage in mice. Strain B originally isolated from a human patient had unchanged virulence for mice after several years in culture. The former strain appeared to be equally virulent for old or young animals, but the latter strain caused a greater mortality in young mice, and the death rate was influenced by the size

of inoculum. Differences in susceptiblity to both strains of *T. cruzi* were noted in mice of different origin, but both A and B strains responded equally well to certain chemotherapeutic agents. Some inconclusive evidence was obtained that the sexes possessed different susceptibilities to the infection.

In assessing results of drug activity, data obtained in experiments in which a survival rate of more than 15 per cent. occurred in controls were not utilized. The early treatment of infections described was more helpful in detecting low activity in drugs than if such treatment were delayed. *J. D. Fulton*

JARPA, A., CHRISTEN, R., AGOSIN, M. & PIZZI, T. Quimioterapia de la enfermedad de Chagas experimental. [**Chemotherapy of Experimental Chagas's Disease.**] *Biologica.* Santiago. 1950, July–Dec., Nos. 12/13, 117–42, 4 graphs. [64 refs.]

The English summary appended to the paper is as follows :—

" The authors investigate the action of Aureomycin, TB one (4 acetylamine-benzaldehyde-Thiosemicarbazone), Streptomycin, Chloromycetin, Chloroquine, Quinine Sulfate and a compound formed by Quinine Sulfate-Pentaquine Phosphate upon *Trypanosoma cruzi* in experimental infestations of mice and in artificial cultures.

" The following facts were taken into consideration in order to evaluate the action of the drugs mentioned above : (*a*) parasitemia ; (*b*) time of survival ; (*c*) histopathological study ; (*d*) weight and physical condition of the animals.

" The only drug that had a favourable action on the course of the experimental infestation in mice was the compound Quinine sulfate-Pentaquine Phosphate, which showed a very marked action on the parasitemia. As regards the other drugs, they did not show the slightest influence on the experimental trypanosomiasis ; on the contrary : Aureomycin as well as TB one produced an intensive increase of parasitemia and a marked shortening of survival time.

" None of the drugs employed had any effect upon the vitality or multiplication rate of *T. cruzi* ' in vitro '." [See this *Bulletin*, 1951, v. 48, 720.]

LEISHMANIASIS

FAWDRY, A. L. & MAZHAR, M. **Two Cases of Kala-azar in South Arabia.** [Correspondence.] *Trans. Roy. Soc. Trop. Med. & Hyg.* 1951, Aug., v. 45, No. 1, 138–9.

Not infrequently, kala azar has been diagnosed tentatively on clinical grounds in Aden ; but hitherto, as far as the authors can ascertain, Leishman-Donovan bodies have not been demonstrated unequivocally there.

They now report two cases seen in January 1951 in children of 6 and 4 years respectively who came from areas on or across the Yemen border. Each had been ill for about a year, with fever and swelling of the abdomen. Enlargement of the spleen and liver was present, together with anaemia. The formol gel test was strongly positive. No plasmodia were found.

In the first case, Leishman-Donovan bodies were found abundantly in puncture fluids from iliac crest and spleen : in the second, they were present in moderate numbers in splenic puncture fluid, but scanty in that from the tibia.

The first patient died 6 hours after the first injection of 25 mgm. urea stibamide, the second before treatment started. Autopsy was not permitted.

H. J. O'D. Burke-Gaffney

BEDNÁŘ, B. Diferenciální diagnosa a průkaz viscerální leishmaniasy (kala-azar) při bioptickém vyšetření. [**Differential Diagnosis of Kala Azar by Biopsy of the Spleen.**] *Časopis Lékařů Českých.* Prague. 1951, Aug. 24, v. 90, No. 34, 1019–20.

The English summary appended to the paper is as follows :—

" The chronic form of kala-azar was diagnosed by means of biopsy from the spleen in a male student aged 22. In 1948 the patient had spent a fortnight in Yugoslavia (Dubrovnik) ; this was the only occasion when he had left Czechoslovakia. No acute illness had been noticed, but two years later the patient developed septic fever, anemia and leucopenia with hepatosplenomegaly. The biopsy made during exploratory laparatomy disclosed clusters of characteristic Leishmanias in cells of the spleen pulp ; the parasites were particularly well seen in sections stained by the Papp method for reticulum. Some difficulty may arise in differentiating the Leishmania from Histoplasma, but this is easily overcome."

LEÓN, L. A. Estudio y presentación de un caso grave de leishmaniasis tegumentaria tratado con Repodral. [**A Severe Case of Cutaneous Leishmaniasis ; Treatment by Repodral.**] *Rev. Kuba Med. Trop. y Parasit.* 1951, Mar.–Apr., v. 7, Nos. 3/4, 31–7, 6 figs.

The term " severe " seems hardly adequate to describe the awe-inspiring condition recorded. The patient was a woman of 43 years ; her disease began 13 years before as a small papule the size of a chick-pea on the right foot. After a fortnight this ulcerated to discharge a yellowish liquid ; the ulcer spread and became very painful. It improved after treatment with copal, a resin of *Hymenea coubaril*, a domestic remedy for wounds and sores. Forty days later, when the patient thought that cure was complete, a fresh nodule appeared over the tendo Achillis ; it ulcerated, discharged and crusted over, but fresh nodules appeared in spite of varied forms of treatment and a condition resembling mossy foot resulted (it is well shown in photographs) and extended up the leg. She suffered also with fever, headache, epistaxis and dyspnoea. In two years she had difficulty in breathing and in swallowing and there were spasmodic attacks of coughing ; the voice became harsh and soon the patient could not speak at all and had to make her wants known by signs, and death was thought to be not far off. Diagnoses considered were syphilis (but serological tests were negative), tuberculosis of the throat ; the leg condition had to be distinguished from elephantiasis, nostras and filarial, from yaws, leprosy and blastomycosis. Finally, leishmania were detected and Repodral was started together with supportive treatment. Repodral contains 13·6 per cent. of antimony. It was given in solution intramuscularly, starting with 1·5 cc. and increasing gradually every 3 days to 3·0 cc. and a total of 30 cc. was given. Later the daily dose was reduced to 2 cc. Improvement was observed within a few days of the treatment being begun, but the drug was continued and in 7 months 110 cc. had been injected and recovery seemed to be complete—the voice had returned to normal, the patient could swallow ordinary food and generally felt quite well. *H. Harold Scott*

BUCK, G., COURDURIER, J., DOREL, R. & QUESNEL, J. J. Premier cas de leishmaniose canine à Madagascar. [**First Case of Canine Leishmaniasis in Madagascar.**] *Bull. Soc. Path. Exot.* 1951, v. 44, Nos. 7/8, 428–30.

FEVERS OF THE TYPHUS GROUP

GIROUD, P., CIACCIO, J. & BABLET, J. Réaction cutanée locale du virus typhique chez un animal qui ne permet pas cette localisation. [**Local Skin Reaction Caused by Rickettsiae in Animals which do not Ordinarily Produce it.**] *C. R. Soc. Biol.* 1951, Apr., v. 145, Nos. 7/8, 570–71.

When guineapigs were inoculated first with a virus derived from a spontaneous pneumonitis of guineapigs and then with rickettsiae intradermally there was no difference in the local reaction or the antibody titres. When, however, the organs of these animals were passaged in rabbits detectable antibodies were present only in those receiving the organs of doubly infected guineapigs, indicating that these were more heavily infected than those having received rickettsiae only.

The authors pursued their studies in 22 rabbits. Adult rabbits localize rickettsiae when these are injected intradermally, producing a local reaction proportionate to the dose and corresponding antibody formation. When, however, a constant dose of "*virus de pneumopathie*" is mixed with a varying dose of rickettsiae intradermal injection of the mixture gives a reaction feebler than that produced by the rickettsiae alone.

On the other hand, small or medium rabbits with thin skins do not localize rickettsiae: although there is congestion and diffuse infiltration, no nodules are formed. Similarly, young rabbits receiving "*virus de pneumopathie*" alone, show no naked-eye reaction, though there is microscopically a light infiltration of the dermis.

When, however, a mixture of "*virus de pneumopathie*" and rickettsiae is injected intradermally into young rabbits, the epidermis is modified, and there is a diffuse dermal infiltration with nodules, endothelial reaction, capillary ectasis and a zone of eosinophilic reaction. Evidently the mixture produces localization, while either organism alone does not.

The authors suggest that by such a means intradermal tests may be established in the case of viruses which diffuse and do not therefore give an evident local reaction. *H. J. O'D. Burke-Gaffney*

BORELL, I. R. Eine Fleckfieber-Epidemie in Barcelona in den Jahren 1942–43. [**An Epidemic of Typhus Fever in Barcelona in 1942–43.**] *Ztschr. f. Hyg. u. Infektionskr.* 1951, v. 133, No. 1, 65–82, 3 figs.

The 1942–43 epidemic of louse-borne typhus in Barcelona is clearly described. Although typhus had long been endemic in Spain there were surprisingly few cases during the civil war of 1936–39. At the end of 1939 endemic foci of the disease existed in Andalusia and Central Spain; great movements of population were occurring, there was great poverty and overcrowding and a complete breakdown of the public health services. In addition to the local foci of the disease there were possible sources of infection from the numerous volunteers who had entered Spain from Russia, Poland, North Africa and other countries in which the disease had been prevalent in recent years. Small outbreaks occurred in many places in Spain in 1940; in 1941 more than 4,000 cases occurred in Malaga and 1,221 cases were reported in Madrid. A few sporadic cases began to occur in Barcelona from June, 1941, but till December, when 14 cases were reported, the average monthly number of cases was less than 3. From the 2nd week of January, 1942, epidemic conditions rapidly became established so that in one week in February there were 170 cases and even higher figures were recorded in two weeks in March. By the last week of June the

epidemic had almost subsided, only 11 cases being reported. Apart from a small flare up in December, 1942, and January, 1943, there were only a few sporadic cases.

The total number of persons attacked was 2,153 in Barcelona city.

The lethality rate was 21·6 per cent. in males and only 11·49 per cent. in females though the age distribution in the sexes was much the same. The greatest discrepancy in the lethality rate according to sex was in the 40 to 50 age-group in which, among 183 males there were 60 deaths and among 162 females there were only 13 ; under the age of 30 the rate was slightly higher among female patients.

The influence of age on the lethality rate was of the usual kind ; in successive decennial periods the rates were, 1·56 ; 1·26 ; 4·49 ; 11·63 ; 21·16 ; 43·41 ; and in persons over 60, 58·79 per cent.

The clinical features were of the usual types. The average leucocyte count was 13,730 per cmm. in 20 fatal cases ; in severe cases it was 8,911 and in mild cases 7,230, but the range of variation in each group was great. Monocytosis was not a special feature ; only 10 out of 112 patients had more than 8 per cent. High myelocyte and metamyelocyte counts were four times more frequent among fatal and severe cases than among mild ones.

The Weil-Felix test was found very helpful though completely negative reactions occurred in 9·1 per cent. of patients who survived and among 28·8 of those who died. High-titre reactions of 1–1,280 were equally common (about 23 per cent.) among fatal and non-fatal cases. In children the titres tended to be lower than in adults. *John W. D. Megaw*

MIKULASZEK, E. & WOJCIECHOWSKI, E. Badania doświadczalne nad budowa antygenowa *R. prowazeki*. Doniesienie I. [**Experimental Studies on the Antigenic Structure of** *R. prowazeki*. **Report I.**] *Med. Dośw. i Mikrob.* Warsaw. 1951, v. 3, No. 2, 141–8.

The English summary appended to the paper is as follows :—

"Chemical fractionation of a purified suspension of *Rickettsia prowazeki* grown in the yolk sacs of embryonated hen's eggs yields 3 cellular fractions. These fractions, based on their behaviour and on some chemical tests, were named : nucleoproteins, polysaccharide symplex and polysaccharide from extracted cells. In complement-fixation tests against sera from human reconvalescents and from rabbits immunized with *R. prowazeki*, these fractions acted as antigens. The most active was the nucleoprotein-fraction (active in dilutions up to 1 : 80,000), the other fractions (symplex and residual polysaccharide) gave a weaker complement-fixation (in dilutions 1 : 6,000 to 1 : 8,000). The isolated fractions did not fix complement in the presence of sera from convalescent guinea-pigs and a rabbit-immune serum for Proteus X19."

BUCCO, G. Ricerche istologiche sulle lesioni meningee nel dermotifo. [**A Histological Study of the Meningeal Lesions of Typhus Fever.**] *Acta Med. Italica.* 1951, June, v. 6, No. 6, 156–64, 7 figs. [15 refs.] English summary.

The author reviews the literature dealing with the pathological changes observed in the meninges in typhus fever and with the various theories propounded as explanations of these changes ; he then describes the naked-eye and microscopic anatomy of the meninges, based on his own studies of the wealth of material available during the Naples epidemic of 1943–44. The condition was found to be one of serous meningitis, characterized by an increase in the

volume of the cerebrospinal fluid. The chief histological features were in the capillaries of the choroid plexus, which were dilated and showed necrotic changes in the endothelial cells ; there was a moderate degree of infiltration of the vessel walls with lymphocytes and other mononuclear cells.

The author concludes that the condition is one of reaction by the choroidomeningeal tissues to allergic "shock" resulting from the interaction of the antigens and antibodies of the disease. Seven photomicrographs illustrate the findings. *John W. D. Megaw*

RÓZNIECKA, D. Badania z zakresu chemoterapii doświadczalnych rickettsjoz. [**Studies of the Chemotherapy of Experimental Rickettsioses.**] *Med. Dośw. i Mikrob.* Warsaw. 1951, v. 3, No. 2, 149–57. [21 refs.]

The English summary appended to the paper is as follows :—

"There was examined the influence of natrium salt compounds of p-aminobenzoic acid, T2, T40, T60, N6, N9 and N11 on intranasal infection with *R. prowazeki* in mice.

"Compounds were administered in a dose of 0·5 mg per gram of weight twice daily during the first three days of illness. Mice treated with compounds have died away later than those not treated. The longest period of illness has been observed after application of nitro-compounds N6, N9 and N11.

"The treatment with T2, T40 and T60 has prolonged the duration of illness for a period of seven days. There was observed a slight effect only after a natrium salt of p-aminobenzoic acid was administered."

MARMION, B. P., MACCALLUM, F. O., ROWLANDS, A. & THIEL, C. C., with technical assistance from J. R. MCDONALD & P. H. PHIPPS. **The Effect of Pasteurization on Milk containing** *Rickettsia burneti. Monthly Bull. Ministry of Health & Pub. Health Lab. Service (directed by Med. Res. Council).* 1951, May, v. 10, 119–28. [14 refs.]

Samples of milk from a herd known to be infected with *R. burneti* and of naturally infected milk to which yolk sac suspensions of the organism had been added, were exposed for periods of 15 seconds in a small H.T.S.T. machine to various temperatures ranging from 154°F. to 163°F. Two experiments were done with infected milk as collected from the farm, two with infected milk to which had been added the previously stored milk of two infected cows from the same herd, and three with infected milk and added yolk sac suspension. For each experiment a total of 30 gallons of milk was available. The day's run through the H.T.S.T. machine was started at 163°–163·5°F. and the temperature was then progressively lowered until a series of samples had been collected which had been subjected to each of the chosen temperature ranges between 154° and 163°F. The plant was then sterilized.

The precise working of the plant and the methods of collection of the milk samples are described. The samples were tested for *R. burneti* by guineapig inoculation. The guineapigs were killed after one month and sera tested in parallel with pre-inoculation samples for complement-fixing antibodies to *R. burneti*. The rickettsial content of the unheated milks was titrated by inoculation of serial tenfold dilutions into guineapigs.

The results of these experiments are tabulated. It was found that naturally infected milk was no longer infective to guineapigs after exposure to a temperature of 154°–156°F. for 15 seconds. With two milks the infectivity of which had been increased by the addition of yolk sac suspensions, guineapigs were consistently infected after heat-treatment at 158°–159°F. and possibly even with milk heated to 160°–161°F. In the latter experiments, phosphatase-negative

milk was still infective to guineapigs. Titration of the artificially infected milks showed them to be infective to guineapigs up to at least a dilution of 1 in 100,000. This concentration of rickettsiae might be equalled by that of milk from individual infected cows but was unlikely in bulked milk.

A small number of experiments on "holder" type pasteurization were also done in the laboratory. Milk containing about $0{\cdot}3 \times 10^4$ guineapig infective doses of *R. burneti* per ml. was exposed to temperatures of 143·5° to 148°F. for 30 minutes, and was no longer infective after exposure within this range; it was still infective after holding at 142·7°F. for 30 minutes. In a second experiment, milk with only one-tenth of this inoculum of rickettsiae was non-infective after exposure to 141·9°F. for 30 minutes.

The authors consider as a result of these experiments that *R. burneti* is rather more heat-resistant than the tubercle bacillus and that the current H.T.S.T. process used in this country does not offer as wide a margin of safety against Q fever as against the risk of tuberculosis. *B. Moore*

DE PRADA, J., GIL COLLADO, J. & MINGO ALSINA, J. L. Tres ixodidos Españoles, vectores de enfermedades rickettsiales. [**Three Spanish Ixodid Ticks, Vectors of Rickettsial Diseases.**] *Trabajos de la Jefatura Provincial de Sanidad de Burgos.* 1951, Mar., 35 pp., 12 figs. [17 refs.]

The three ticks in question are *Hyalomma savignyi*, *H. excavatum* and *Rhipicephalus pusillus*. The first two are vectors in Spain of Q fever. The bite of *Rhipicephalus pusillus* has been found to produce headache, adenitis and inguinal swelling after 13 days. The authors ascribe this to an unknown rickettsial disease. Infected persons show no reaction with *Proteus X19* or with any of the antigens of Q fever, and the disease is described as a member of the spotted fever group. *J. H. Sudd*

GIROUD, P., JADIN, J. & JEZIERSKI, A. Le pou peut-il jouer un rôle dans la transmission de la fièvre Q ? [**Can the Louse Play a Part in the Transmission of Q Fever ?**] *C. R. Soc. Biol.* 1951, Apr., v. 145, Nos. 7/8, 569–70.

The authors refer to their previous identification, by guineapig inoculation, of *R. burneti* infection in human body lice in Ruanda-Urundi some 3 months, after a local epidemic of Q fever had occurred [this *Bulletin*, 1951, v. 48, 545].

They have now confirmed these findings by feeding 250 fresh lice on guineapigs and young mice after these animals had been inoculated with a highly virulent strain of *R. burneti*. The lice were fed on these animals for two successive days and then, after one day's fast, on a normal monkey.

The lice rapidly became red and all died 5–6 days after their meal. The dead, and later the living, lice were inoculated into guineapigs 5 days after the infected meal. The guineapigs reacted, after variable or almost no periods of incubation, with temperatures of 40 to 41°C.; their spleens were enlarged and full of rickettsiae. Peritoneal adhesions were constant. Those which died had no detectable antibodies, but the infection was confirmed by animal passage.

The most important feature was, however, that the dejecta of the lice were infective 3 days after the meal. Their virulence was less than that of whole lice, but nevertheless typical infections were produced in guineapigs inoculated with them and agglutinins appeared on the 16th day in titres of 1/80: rickettsiae were easily detected in the spleens of the infected guineapigs.

It is evident, as *R. burneti* is very resistant to external agents, that the rôle of lice on highly infected persons is very important, since the dejecta of such lice may be infective. *H. J. O'D. Burke-Gaffney*

PIZZILLO, G. Sulla febbre esantematica mediterranea. [**Mediterranean Exanthematic Fever.**] *Acta Med. Italica.* 1951, June, v. 6, No. 6, 149–55, 3 figs. & 1 graph. [24 refs.]

The author begins by reviewing the literature dealing with the tick-borne fevers of the typhus group to which " a myriad " of names have been applied by different writers. The information supplied is interesting but not always reliable ; he includes, for example, tsutsugamushi fever in the list of names quoted and attributes the name "*tifo da zecca*" [tick typhus] to CASTELLANI and JACOMO (1937).

A case is described in which a young man of 20 years found a tick, assumed to be *Rhipicephalus sanguineus*, firmly attached and needing considerable force for its removal ; the tick was believed to have come from a dog beside which he lay during a journey in a crowded train from Messina to Palermo. Twenty-one days later he had an attack of severe typhus-like fever which lasted 14 days. The onset was sudden, the fever was of a high continued type, ending by crisis. The rash was observed on the first day, it became maculopapular with a tendency to be petechial and it had almost faded by the end of the attack. The long incubation period was regarded as an extraordinary circumstance though, curiously enough, it was exactly the same as in the personal attack following a tick bite, which prompted the reviewer 30 years ago to suggest the name tick typhus as suitable for all the tick-borne typhus-like fevers.

The special treatment adopted by the author was daily intravenous injections of vitamin C and adrenaline, given from the third day until the end of the fever, but the temperature chart does not suggest that the author is justified in claiming brilliant results from the synergic action of these drugs.

A remarkable circumstance was that ten hours after the forcible removal of the tick the patient was attacked by high intermittent fever with arthritis lasting 13 days and followed by a period of convalescence of 7 days before the onset of the attack of tick typhus.

[The author has been badly served by the translator of the summary in English ; the opening sentence reads : " L'A. exposes the mediterranian fever and talks about it in the chapter on the ' thifoid fever by ' " ; presumably the word tick has been omitted at the end. Even more enigmatic is " the static poof of the rod " as a translation of the Italian "*prova statica del laccio*" which refers to method of making a latent rash visible by constricting a limb.]

John W. D. Megaw

YELLOW FEVER

LUMSDEN, W. H. R. & BUXTON, A. P. **A Study of the Epidemiology of Yellow Fever in West Nile District, Uganda.** *Trans. Roy. Soc. Trop. Med. & Hyg.* 1951, Aug., v. 45, No. 1, 53–78, 5 figs. [32 refs.]

Much of the earlier work published from the institute at Entebbe, under the Rockefeller Foundation, dealt with the epidemiology of the yellow fever in an area of equatorial forest at Bwamba, Western Uganda. In that place, the adult *Aëdes africanus* was always present, so that the chain of infection from mosquito to monkey may well have been maintained throughout the year.

The authors of the present paper have turned their attention to an area with a pronounced dry season in the West Nile District, Uganda. It would be supposed that the cycle of transmission cannot be so easily maintained, because the breeding places of many of the mosquitoes dry up for a part of the year.

The paper forms an excellent full-length introductory study : the authors have evidently read a wide range of papers and give sufficient description of topography, geology, climate and vegetation. The area, of about 4,000 sq. miles, has a population of about a quarter of a million people, mainly agricultural. A great variety of crops are grown, but none of them is likely to provide mosquito breeding places, except certain varieties of banana, and they are not common.

The earlier work and the present survey have demonstrated that a small proportion of the people are immune to yellow fever, though there are parts of the area where all the individuals tested were negative ; in general, the rate of immunity is less than in surrounding areas. The present authors have examined a total of 350 people, using the mouse protection test, and consider their data fully. The proportion of immunes is higher in males than in females to an extent which is just significant statistically. It is observed that the higher rates tend to occur in places near forests, but persons with immunity are sporadically distributed in many localities.

A considerable number of animals were shot and the blood tested for immune bodies. Among the adult monkeys, 7 out of 14 *Colobus abyssinicus ituricus* were positive, and one out of 6 *Cercopithecus aethiops centralis*. The two baboons (*Papio doguera tessellatus*) were both negative. Among the other animals examined, was a single red river hog (*Potamochoerus porcus*), which was positive : it had previously been shown that antibody develops in this animal after inoculation with the yellow fever virus. Among 14 other animals (bush babies, hares, rock hyrax, etc.) none was found showing immunity.

As to mosquitoes, over 40 species were taken ; catches were made at a number of places throughout the 24 hours, African children being exposed at ground level and on platforms on trees. At the end of the dry season, practically all the adult mosquitoes belonged to species which would breed in pools in river beds and such places, hardly any of them having been bred in rot holes or other "containers". The data concerning mosquitoes are tabulated in relation to height from ground, and time of day or night. Taking the figures for all species together, 38 mosquitoes bit by day, 475 by night : of those biting by night, attack was most frequent in the period just after sunset, but in *A. gambiae* and *funestus*, the main peak was just before dawn.

In early March, at the end of the dry season, all the tree holes were dry. An inch of rain fell, after which many of the holes contained water ; about half of them also contained larvae, identified as those of 5 species of *Aëdes* and *Culex horridus*. Dry débris was scraped from about 100 holes and soaked : small numbers of larvae of *A. ingrami*, *simpsoni* and of some member of the *africanus* group emerged from the material. A very few larvae were found in the axils of certain wild plants. In certain types of cultivated banana, the axil contains water, but, though 150 of these were examined, only one mosquito larva was found. Larvae were not found in a village in waterpots and domestic utensils.

The paper terminates with an interesting discussion on the interpretation of the facts and the probable seasonal incidence of the transmission of yellow fever. [We look forward to more work from other areas which have a seasonal drought, but we hope that the authors will not often refer to "adults ecloding from pupae".]

P. A. Buxton

RABIES

LÉPINE, P. & CROISSANT, O. Microscopie électronique des corps de Negri dans la rage des rues. Méthode de repérage et d'examen des coupes histologiques en microscopie électronique. [**Electron Microscope Studies of Negri Bodies in Rabies.**] *Ann. Inst. Pasteur.* 1951, July, v. 81, No. 1, 1–8, 1 text fig. & 10 figs. on 2 pls. [13 refs.]

Serial sections of rabies virus-infected brain are observed under the light microscope. If an interesting region containing Negri bodies is discovered this is outlined on the slide and the corresponding area subsequently marked with a needle on the paraffin block from which it is cut out in the form of a pyramid. The paraffin is dissolved in toluol and the block immersed in monomeric butyl metacrylate, which is later polymerized in the drying chamber in gelatin tubes according to the technique of NEWMAN, BORYSKO and SWERDLOW. The resulting plexiglass cylinder is then cut in a Spencer microtome equipped with a thermic advancement installation developed by the above-mentioned authors. The classical razor blade is replaced by a sharp-edged piece of glass. The tissue section is placed on formvar [a polyvinyl formaldehyde plastic] floating on the surface of water. With the help of micro-manipulation and phase microscopy the part to be examined is placed in the centre of a wire mesh screen. The preparation is then dried and immersed for at least 24 hours in a solvent and subsequently shadowed with metal vapour.

Reproductions are appended to the article showing the same group of cells and Negri bodies under the light, the phase and the electron microscope.

The details of the structure of the Negri bodies are described, and contrary to expectations and to earlier results obtained with Guarnieri and Bollinger bodies, they do not show elementary bodies of a size corresponding with that of the virus calculated by a number of indirect methods. The opaque bodies seen measure between 320 and 580 millimicrons in diameter. They, as well as the corresponding holes or niches, would each be able to accommodate 3–4 viruses.

Negri bodies are described as coagulated "*plasma cellulaire*" surrounding a group of virulent elementary bodies or as virus masked with cellular antibodies. This interpretation conforms with results of previous histochemical studies which demonstrated that only the internal structures of Negri bodies stain like nucleic acid. *C. Klimt*

REMLINGER, P. & BAILLY, J. Action de l'auréomycine sur le virus rabique. [**Action of Aureomycin on Rabies Virus.**] *Ann. Inst. Pasteur.* 1951, July, v. 81, No. 1, 83–4.

Amorphous and crystalline aureomycin, in 1 per cent. solution, inactivates fixed rabies virus *in vitro* after a minimum contact period in the dark of 6 hours at 6°C. *C. Klimt*

SHIBUKI, M. Activité du vaccin formolé antirabique en comparaison de celle du vaccin phéniqué. [**Activity of Formolized Antirabic Vaccine Compared with Phenolized Vaccine.**] *Japanese J. Exper. Med.* 1950, Sept. 30, v. 20, No. 6, 699–703.

The vaccines tested were prepared from guineapig brain inoculated with virus passaged in guineapigs. Ten per cent. brain suspensions were centrifuged and the supernatant was divided into two equal parts. One part was inactivated with 0·25 per cent. formalin for 3 days at ice-box temperature. The other part was treated with 0·5 per cent. phenol and incubated for 48 hours at 37°C.

Experiment 1. Groups of mice were immunized intraperitoneally with 3 doses of 0·25 cc. of various dilutions of formalin-inactivated vaccine given at 2-day intervals and then challenged intracerebrally with 50–500 LD_{50} of the homologous virus. Immunity was detectable as early as 2 days after the last injection and reached its maximum 5 days later.

Experiment 2. When only one dose of 0·5 cc. of undiluted formalin-inactivated vaccine was given, maximum immunity was reached after 2 weeks, at which time protection was afforded against 1,600 LD_{50}.

Experiment 3. Comparison was made of formolized and phenolized vaccine prepared in the manner described above from a common virus source. Percentage survival rates upon challenge with a constant amount of 100 LD_{50} of homologous virus at weekly intervals showed a superiority on average in favour of the formalin-inactivated vaccine, although not consistently so when compared week by week. [The conclusions of the author regarding the superiority of formolized over phenolized vaccine does not, on the basis of his own figures, seem warranted. No comparison on the basis of titrations of LD_{50} protection values was carried out for this purpose.] *C. Klimt*

PLAGUE

LINK, V. B. **Plague.** *Amer. J. Trop. Med.* 1951, July, v. 31, No. 4, 452–7, 2 maps & 2 figs.

This is a very brief review of existing knowledge of plague, with special reference to its occurrence in the Western States of the U.S.A. The production of the article has been prompted by a recent increase in the number of cases of the disease ; between July, 1949 and July, 1950, there have been 6 confirmed cases in New Mexico and Arizona and 10 unconfirmed cases in 5 Western States. Plague-infected rodents have also been found in 16 additional counties during 1949 and 1950, so that now wild-rodent infection has been demonstrated in 138 counties of 15 Western States.

During the past 25 years no case of plague in man has been caused in the U.S.A. by transmission from domestic rodents, so that at present attention is being directed specially to the prevention, diagnosis and treatment of sporadic cases resulting from association with infected wild rodents. The only practicable means of prevention is avoidance of contact with the rodents. Diagnosis in bubonic cases is by finding the bacilli in stained smears of fluid aspirated from the buboes. In septic and pneumonic cases blood and sputum smears are examined. Cultural and animal-inoculation tests give delayed results and therefore do not help in supplying the immediate diagnosis that is essential as a guide to early treatment by streptomycin and sulphonamides, which, given together, yield excellent results. In all doubtful cases treatment should be started at once. Streptomycin is given in doses of 0·25 to 1·0 gm. every 4 hours till defervescence; sulphadiazine is given in an initial dose of 4·0 gm. followed by 1·5 to 2 gm. every 4 hours and is continued for 10 to 15 days after defervescence.

John W. D. Megaw

AMIES, C. R. **The Envelope Substance of** *Pasteurella pestis.* *Brit. J. Exper. Path.* 1951, June, v. 32, No. 3, 259–73, 8 figs. on 2 pls. [28 refs.]

" The so-called envelope of *Past. pestis* is nothing more than a particularly well-developed bacterial capsule. This conclusion was reached by studying the capsule in wet Indian ink preparations illuminated by a dark-field condenser (Rowland's method).

" The capsular substance can be removed from the cells by certain aqueous solvents such as potassium thiocyanate. The one important immunogenic-antigen of *Past. pestis* is thus obtained in a soluble form, and it can be concentrated and purified by the usual physico-chemical methods.

" The final product appears to be a simple protein. It is strongly antigenic, a few micrograms being sufficient to induce a high level of immunity in experimental animals. The chemical characterization of the purified antigen has, however, not yet been determined, and until this is done no claims can be made regarding the degree of purity achieved.

" The capsules of the pneumococcus and *K. pneumoniae*, which are composed of polysaccharide, are not soluble in potassium thiocyanate, whereas the polypeptide capsule of *B. mycoides* can be extracted by this method.

" The methods employed hold promise of wider application in the field of immunochemistry and bacterial technology."

CHOLERA

PANDIT, C. G. & HORA, S. L. **The Probable Role of the Hilsa Fish, Hilsa Ilisha (Ham) in maintaining Cholera Endemicity in India.** *Indian J. Med. Sci.* 1951, Aug., v. 5, No. 8, 343–56, 6 figs.

No answer has yet been obtained to the question how the cholera vibrio persists in inter-epidemic periods. The evidence so far is that the carrier state, either in convalescents or contacts, is of short duration and it has not been found that sub-clinical infection exists in inter-epidemic periods. The vibrio has not been found in open water sources such as tanks, canals and rivers, except in the presence of cholera in the vicinity. In view of certain findings reported by Japanese workers the OIHP/WHO study group on cholera had recommended the investigations of possible persistence in fish and other aquatic fauna. This question was taken up by the authors.

A comparison of maps of the distribution of endemic cholera areas in relation to river systems showed that these were areas in which Hilsa fisheries were located, and it was considered suitable to investigate the possible rôle of Hilsa as a reservoir of infection.

The authors first bring out the facts in relation to each of the main river systems and note that the endemic areas are related to those flowing into the Bay of Bengal and not those of the West Coast running into the Arabian Sea. The point is later brought out that the Arabian Sea is, generally, of higher salinity. The Hilsa is essentially a brackish water species, migrating up the rivers at certain periods of flood. Their bionomics and movement at different stages of their development and at different seasons are discussed. The possibility is considered that the young Hilsa moving up river near the bottom, where they will be away from the sun's rays, may be a reservoir of infection. Factors affecting the salinity of the mouths of the Ganges and its estuary, at different seasons, are dealt with and its effect on the physiology of the fish and its effect on the vibrio are considered to be worthy of study.

Reference is made to Russell's observations on relationship between the incidence of cholera and rainfall and humidity. The authors remark—" It may be that the correlation which has been observed between these factors and cholera incidence is there, because of the influence of these factors on the bionomics of the fish". Another point mentioned is a possible relationship

between a five-year cycle of cholera in Eastern Bengal and a five-year cycle of the Hilsa fishing. The distribution of Hilsa species in other cholera and non-cholera areas and their possible migrations are discussed shortly.

No direct field or laboratory studies on the matter have so far been undertaken in India and it is recognized that these, including experimental infection of fish, will be necessary before any evidence of value on the matter can be obtained. "It is felt that, at any rate, a *prima facie* case has been made for such studies."

J. Taylor

AMOEBIASIS AND INTESTINAL PROTOZOAL INFECTIONS

BONILLA NAAR, A. & RODRÍGUEZ VELANDIA, Olga. Amibiasis autóctona en Bogotá (2,640 Mts.) e incidencia de parásitos intestinales en la clase media. (Parasitismo intestinal y eosinofilia.) [**Autochthonous Amoebiasis in Bogotá and Intestinal Parasitism in the Middle Classes. Eosinophilia and Intestinal Parasitism.**] *Rev. Kuba Med. Trop. y Parasit.* 1951, Mar.–Apr., v. 7, Nos. 3/4, 52–3.

By "middle class" the authors seem to imply those attending a private laboratory or clinic and not the ordinary hospital patient. Their returns are based on examination of 4,051 cases at Dr. de Castro's private laboratory and 200 clinical histories of patients attending the clinic of Dr. Bonilla Naar.

Of protozoal infections the following percentages were observed : *Entamoeba histolytica* 4·5, *E. coli* 9, *Iodamoeba bütschlii* 4, *Giardia intestinalis* 0·5, *Chilomastix mesnili* 0·5. Other investigators have returned much higher percentages [from examination of all classes in Bogotá, autochthonous and introduced ?] ; up to 21·5 for *E. histolytica*, 17·5 for *E. coli*, 9·5 for *Giardia intestinalis*, 6·3 for *Iodamoeba bütschlii*. As regards helminthic infestations the authors record *Trichuris* 19·5 (44), *Ascaris lumbricoides* 10 (28·5), hookworms 1 (23·5), *Strongyloides* 1 (2·2), *Enterobius vermicularis* 0·01 (0·07) ; the figures in brackets are the maximal percentages recorded by others.

Next, eosinophilia and parasitism. In 100 clinical histories investigated 79 per cent. of those parasitized showed eosinophilia ; most marked in those infested with *Ascaris*, but in one patient with *Trichuris* of average intensity there was a 27 per cent. eosinophilia ; in 11 per cent. parasites were present without eosinophilia and in 7 per cent. the reverse was seen—eosinophilia but no parasites. Of those with a combination of parasites 35 per cent. had eosinophilia up to 30 per cent., while 3 had no eosinophilia. [No other details are given as regards the combination of parasites or the degrees of eosinophilia.]

H. Harold Scott

PHILLIPS, B. P. **Comparative Effects of certain Species of Trypanosomidae on the Growth of *Endamoeba histolytica* in the Absence of Bacteria.** *Amer. J. Trop. Med.* 1951, May, v. 31, No. 3, 290–94, 1 fig.

In previous studies [this *Bulletin*, 1950, v. 47, 624, 741] it has been demonstrated that *Entamoeba histolytica* can be grown in pure culture with *Trypanosoma cruzi*. In the present paper the author describes further observations on the growth of the amoeba with *T. cruzi*, *T. conorhini*, *T. pipistrelli*, *Leishmania donovani*, *L. tropica*, *L. brasiliensis* and *Strigomonas fasciculata*.

Stock cultures of the flagellates were maintained in a medium consisting of a base (Bacto-agar 2·4 per cent., trypticase 2·0 per cent., NaCl 0·5 per cent., and defibrinated rabbit blood 10 per cent.) and overlay (trypticase 1·0 per cent., NaCl 0·8 per cent. and dextrose 0·5 per cent.). For growth with the

amoeba, the flagellates were kept in a liquid medium composed of trypticase 2·0 per cent., NaCl 0·7 per cent., Na_2CO_3 0·03 per cent., dextrose 0·5 per cent. and horse serum 4·0 per cent. Both types of cultures were incubated at 24°C. For the amoeba-flagellate culture, grown in test-tubes and microtubes, 4 parts of the flagellate culture were mixed with 1 part of a 0·3 per cent. solution of sodium thioglycollate in saline. Each microtube culture was inoculated with a single amoeba isolated with the help of a micromanipulator. The mixed cultures were incubated at 37°C.

The results obtained with the different flagellates were as follows. In microtubes growth was good with *T. cruzi*, fair with *S. fasciculata*, and very poor with the remaining Trypanosomidae. In test-tubes the amoebae grew well through indefinite numbers of subcultures with *T. cruzi*, but could not be maintained serially with any of the other flagellates. In cultures the amoebae were observed to ingest the associated flagellates. *C. A. Hoare*

NAKAMURA, M. & ANDERSON, H. H. **Evidence of Direct Amebacidal Action of Agents tested** *in Vitro* **against** *Endamoeba histolytica.* *J. Parasitology.* 1951, Aug., v. 37, No. 4, 421–3, 1 fig.

In testing potential amoebicidal action *in vitro* in the presence of one or more bacteria it has been recognized that the agents used may act indirectly on the amoebae through affecting the bacteria. Since PHILLIPS [this *Bulletin*, 1950, v. 47, 624] has been able to culture *E. histolytica* in presence of *T. cruzi* without bacteria it appears that an advance in the technique of *in vitro* testing has been made, because this trypanosome is resistant to treatment with practically all known therapeutic agents. The criteria used by the present authors during tests of some accepted amoebicides and antibiotics (carbarsone, emetine, aureomycin, bacitracin, polymyxin B, and terramycin) to indicate that *T. cruzi* was unaffected by these substances at amoebicidal concentrations, were motility and growth on subculture. A third criterion, namely absence of inhibition of respiration of the trypanosomes in the presence of amoebicidal concentrations of the drugs, has now been suggested. The measurements of O_2 uptake were made on conventional apparatus in an atmosphere of air at 37·2°C. It was found that the drugs named did not affect oxygen uptake at levels where amoebicidal action was apparent. This was accepted as additional proof that the effect of these substances on *E. histolytica* was direct.

J. D. Fulton

CHAKRAVARTY, A. **Pulmonary Amoebiasis.** *J. Indian Med. Ass.* 1951, Aug., v. 20, No. 11, 399–402, 6 figs. on 3 pls.

From the clinical aspect pulmonary amoebiasis may be divided into : (1) *Symptomatic.* In this group no involvement of lung tissue can be ascertained radiologically. The clinical manifestations appear to be due to involvement of the diaphragmatic pleura which is supplied by phrenic nerve and lower six intercostal nerves. (2) *Secondary pulmonary amoebiasis* in which there is actual invasion of lung tissue from infected liver. (3) *Primary pulmonary amoebiasis,* which indicates pulmonary involvement without amoebic hepatitis.

During two years 6 cases were collected by the author in India. In the first three, diagnosis was confirmed by the demonstration of *E. histolytica* in the sputum.

In the sixth, though *E. histolytica* was found in the sputum, there was no evidence of an abscess cavity in the lung, but the infiltrational lesions in the subapical- and mid-zone were probably tuberculous in origin.

In pulmonary amoebiasis the examination of faeces occupies a very limited place. A positive result is suggestive, but not decisive.

The paper is illustrated by 8 skiagrams. *Philip Manson-Bahr*

MILLER, M. J. & GILANI, A. **The Clinical Significance of Non-Dysenteric Intestinal Amoebiasis.** *Trans. Roy. Soc. Trop. Med. & Hyg.* 1951, Aug., v. 45, No. 1, 131–6.

Of at least 200,000,000 people throughout the world who are infected with *Entamoeba histolytica* only a small percentage show signs and symptoms of amoebic dysentery, and there is another small group who are convalescent carriers. It is claimed that in the majority signs and symptoms of amoebic dysentery never did manifest themselves, and probably never will. The term "non-dysenteric intestinal amoebiasis" is the rather clumsy term proposed for the enigmatical condition. The causal relationship of the psychological phenomena often described in association with intestinal amoebiasis has never been established. In Calcutta the number of patients admitted with a presumptive diagnosis of chronic amoebiasis comprised 5 to 10 per cent. of all admissions during the period of observation. The symptom-complex presented by these patients was predominantly referable to the gastro-intestinal tract, but a readily diagnosible clinical entity was not apparent. A total of 36 patients were studied, all males over 13 years of age. Of these 36 there were 9 positive and 27 negative for *E. histolytica* infection. All these received amoebicidal drugs.

The results of this study go to show that a group of patients with chronic gastro-intestinal complaints and infected with *E. histolytica* did not show a response to therapy with amoebicidal drugs any better than a similar uninfected group treated symptomatically. Thus 3 out of 8 in the former group showed definite improvement while 14 of the 27 in the latter group were improved. These results are held to indicate that in the infected group the use of anti-amoebic therapy did not result in a higher cure rate, suggesting that eradication of the parasite did not alter the clinical state of the patient.

Because of the protean symptomatology often ascribed to non-dysenteric intestinal amoebiasis, it becomes important that the precise clinical status of this group of amoebic infections should be clarified. Otherwise the discovery of *E. histolytica* in the stool may divert attention from the true underlying cause of the patient's disease.

Treatment and details of the patients' symptomatology are shown in 3 tables and discussed at length.

[This appears to the reviewer to be an alarming paradox which rules out the value (which can hardly be contradicted) of faeces examination altogether.]

Philip Manson-Bahr

HOEKENGA, M. T. **A Comparison of Aureomycin and Carbarsone in the Treatment of Intestinal Amebiasis.** *Amer. J. Trop. Med.* 1951, July, v. 31, No. 4, 423–5.

Thirty local patients in Honduras, most of them suffering from acute amoebiasis, were treated with one or other of two courses of treatment with aureomycin ; 40 others were treated with carbarsone and sulphadiazine. The aureomycin treatments produced alleviation of the acute symptoms of amoebic dysentery within 24 to 36 hours ; the carbarsone-sulphadiazine treatment did so within 48 hours. The criterion of cure was the absence of parasites on examination of a minimum of six specimens of stool between one week and three months after completion of treatment. One parasitic relapse was observed within two days of the completion of aureomycin treatment, and 50 per cent. of the relapses after this drug were observed within a month. Relapses after the carbarsone-sulphadiazine treatment also tended to occur within this period.

The results are summarized in the following table, which has been drawn up by the author :—

	Number of cases	Number of apparent cures	Per cent. of apparent cures
1. Aureomycin 250 mgm. twice a day for 10 days (total 5·0 gm.)	15	8	53
2. Aureomycin 250 mgm. thrice a day for 10 days (total 7·5 gm.)	15	12	80
3. Carbarsone 250 mgm. thrice a day for 10 days plus Sulphadiazine 3·0 gm. priming dose and 1·0 gm. four times a day for 5 days	40	21	52

He concludes that aureomycin in the larger dosage (totalling 7·5 gm. over 10 days), is an effective amoebicidal agent ; in bigger doses still it would probably be even more effective. *A. R. D. Adams*

TOBIE, J. E., MOST, H., REARDON, Lucy V. & BOZICEVICH, J. **Laboratory Results on the Efficacy of Terramycin, Aureomycin and Bacitracin in the Treatment of Asymptomatic Amebiasis.** *Amer. J. Trop. Med.* 1951, July, v. 31, No. 4, 414–19, 1 fig.

At an institution for mental defectives the incidence of *Entamoeba histolytica* infection was known to be high ; clinically overt manifestations of its presence in the form of amoebic dysentery and of amoebic liver abscess appeared from time to time. The opportunity was taken of examining the efficacy of terramycin, of aureomycin, and of bacitracin in the mass treatment of amoebiasis. For this purpose four " infirmaries " [distinct blocks of building] were selected ; each contained about 200 patients.

An average of four stools from each of a number of randomly selected patients in each block was examined to estimate the incidence of the infection. By this means 49 per cent. of the patients in the block used for terramycin treatment were estimated to be infected before treatment ; 45 per cent. in the aureomycin group ; 68 per cent. in the bacitracin group ; and 64 per cent. in the fourth and untreated control group. All the patients in each block, or group, were given the treatment appropriate to that group, irrespective of whether there was proof of infection or not ; each group was kept isolated during and after treatment to minimize the risk of cross infection and of reinfection. Stools of the sample patients who had had their stools examined before treatment were re-examined at fixed intervals after the completion of treatment ; the conclusions are based on these examinations.

Every patient was treated, coincidentally with all the others, for a period of ten days. The dosage of terramycin was 2 gm. daily for patients over, and 1 gm. daily for those under, 75 lb. body weight ; that of aureomycin was the same ; and that of bacitracin 80,000 units or 40,000 units daily for similar weights. Two weeks after treatment terramycin appeared to be 100 per cent. effective, aureomycin also 100 per cent. effective, but bacitracin only 59 per cent. effective, as judged by the absence of *E. histolytica* from the stools.

Two and a half months after the treatment stool examinations were repeated on the same sample patients ; the figures now were terramycin still 100 per cent., but aureomycin 60 per cent., and bacitracin only 28 per cent. effective.

Six months after treatment every individual (222 patients), and not merely the sample patients (10 in number), in the terramycin group had his stool examined on two occasions. The cure-rate of this drug, as judged by the continued absence of parasites from the ten sample patients, was still 100 per cent. ; but the examination of the remaining 212 patients showed two still to be infected (*i.e.*, 1 per cent.).

The incidence of infection in patients in the untreated control group remained substantially unchanged throughout.

The efficacy of terramycin in sterilizing a bowel infection with *E. histolytica* is thus clear. The authors do not consider that this experiment establishes the superiority of terramycin over aureomycin as a sterilizing agent in amoebiasis. Among other reasons for this conclusion, they point out that the possibility of reinfection in this type of patient cannot satisfactorily be excluded. Bacitracin they consider to be demonstrably inferior as an effective amoebicide both to terramycin and to aureomycin.

There is evidence that terramycin treatment eliminates *Iodamoeba bütschlii* infections, and that it reduces the number of infections with *Entamoeba coli* and with *Endolimax nana*. *A. R. D. Adams*

BASNUEVO, J. G., COWLEY CHÁVEZ, O. & BLANCO RABASSA, E. La cloroquina en el tratamiento de las ulceraciones intestinales por *Endamoeba histolytica* (II). [**Chloroquine in the Treatment of Intestinal Ulcerations by *Endamoeba histolytica* (II).**] *Archivos Hospital Universitario.* 1951, July–Aug., v. 3, No. 4,355–83, 8 figs. on 7 pls. [34 refs.] English summary.

Four cases of amoebiasis and 1 of giardiasis successfully treated.

WAGNER, O. Fortschritte in der Amöbenruhr-Therapie auf biologischen und methodischen Grundlagen. [**Progress in the Experimental Therapy of Amoebic Dysentery from the Biological Standpoint.**] *Ztschr. f. Tropenmed. u. Parasit.* Stuttgart. 1951, June, v. 3, No. 1, 47–65. [Numerous refs.]

In this review article on the progress made during the last 25 years regarding the aetiology, host-parasite relations, pathogenic characters and response to chemotherapy of *E. histolytica*, a wide survey of the literature has been made. The views of different authors on these matters are frequently conflicting. DOBELL in his earlier work, and FAUST, have held that *E. histolytica* is an obligate tissue parasite, while others, including HOARE, and DOBELL in his later work, believe that this organism may live as a commensal in the lumen of the gut. REICHENOW [this *Bulletin*, 1938, v. 35, 580] has maintained that its normal habitat is the lumen and that it assumes invasive characters only under certain conditions. WESTPHAL [*ibid.*, 586] has shown that bacteria by damaging the intestinal wall are in some circumstances the determining factor in the pathogenicity of the organism. The normal cyst-forming *minuta* types are credited with this property in contrast with the larger forms which invade the tissue, and thus a plausible explanation of the existence of healthy carriers is provided. The author goes on to describe the cultural characters of the organism, and the suitability of various media is dealt with, a reference to the work of PHILLIPS [this *Bulletin*, 1950, v. 47, 624] who was able to maintain *E. histolytica* with *T. cruzi* in the absence of bacteria, being included. The present author favours the rat as host for experimental infections when used as described by JONES [*ibid.*, 1947, v. 44,313]. The rabbit, which is not naturally infected with *E. histolytica*, can also be used experimentally provided that the part of the gut functionally similar to the caecum in man be considered. In monkeys the *minuta* forms of the amoebae which give rise to cysts are present, but tissue invading forms which ingest red blood cells are generally absent.

Kittens are not naturally infected, and, as the present author has shown, the accompanying bacteria play an important part in these animals when infected experimentally with amoebae. They do not harbour cysts or respond to emetine and therefore their value as hosts in chemotherapeutic experiments is open to doubt, and the author expresses similar doubts about the guineapig. A brief survey of the types of compound which have proved of value in the treatment of amoebiasis is given in two tables, and includes plant alkaloids, arsenoxides, dithioarsenites, arsinic acids, quinoline derivatives, acridines and antibiotics, including aureomycin, terramycin, bacitracin and others. The bismuth salt of *p*-glycolylaminophenylarsinic acid, also known as Wia, Milibis or Viasept, whose properties have been described in this *Bulletin* [for example, 1951, v. 48, 154] is relatively non-toxic and has been used prophylactically.

J. D. Fulton

SADUN, E. H., BRADIN, J. L., Jr. & FAUST, E. C. **The Effect of Ascorbic Acid Deficiency on the Resistance of Guinea-Pigs to Infection with** *Endamoeba histolytica* **of Human Origin.** *Amer. J. Trop. Med.* 1951, July, v. 31, No. 4, 426–37. [21 refs.]

Experimental and clinical evidence in man and experimental animals has pointed to the fact that the course of amoebic infection is influenced markedly by diet [this *Bulletin*, 1935, v. 32, 191 ; 1936, v. 33, 528, 547 ; 1950, v. 47, 538] either favourably or unfavourably. SADUN *et al.* [this *Bulletin*, 1950, v. 47, 852] found that the guineapig, apparently the only small animal which requires preformed vitamin C in the diet, became very susceptible to infection with *E. histolytica* when given inadequate amounts of this substance. This host, therefore, appeared to be well suited to controlled studies on vitamin C deficiency in relation to resistance to infection with *E. histolytica*. One group of female guineapigs weighing about 220 gm. housed in individual cages was given a purified experimental (scorbutogenic) diet of known composition, which is described, along with unrestricted supplies of water. A second group received in addition 20 mgm. ascorbic acid by mouth on alternate days, an amount which appeared to be satisfactory for the maintenance of normal health. A third group was given a crude diet somewhat deficient in ascorbic acid. The guineapigs were inoculated intracaecally 10 days later with trophozoites of *E. histolytica* and an uninoculated group of animals receiving similar diets served as controls. The experiment lasted 30 days and during this period each animal was weighed at 5-day intervals and the ascorbic acid content of plasma, liver, spleen, and muscle was determined in a few animals from each group. Post-mortem examination of the intestine was made at death or when the animals were killed at the end of the experimental period, and the presence or absence of *E. histolytica* in the caecal contents was determined. Certain other organs were also preserved for histological examination. Those animals which received diets deficient in ascorbic acid were found to be more susceptible to infection with *E. histolytica*. All those on the scorbutogenic diet which developed intestinal lesions (86 per cent.) died apparently on account of the infection, whereas only 27 per cent. of deaths occurred in similar circumstances when ascorbic acid was added to the diet. In those receiving the deficient diet the disease was of fulminating character, and in those receiving the less deficient diet mortality was intermediate to those in the other two groups. The authors point out that the deficiency of ascorbic acid does not necessarily mean that this factor *per se* is the cause of decreased resistance to infection, but that the effect may be caused indirectly through resultant lowered food intake, or some other factor which affected the organism as a whole. In infected animals

hepatomegaly and splenomegaly was the rule, but there were no significant alterations in the distribution of ascorbic acid in the tissues. *J. D. Fulton*

REINERTSON, J. W. & THOMPSON, P. E. **Experimental Amebic Hepatitis in Hamsters.** *Proc. Soc. Exper. Biol. & Med.* 1951, Mar., v. 76, No. 3, 518–21, 4 figs.

Using a human strain of *Entamoeba histolytica* growing in culture with 3 species of bacteria (of the coliform, *Proteus* and *Streptococcus faecalis* groups) the authors produced amoebic hepatitis in golden hamsters (*Cricetus auratus*). The best results were obtained with an inoculum of 50,000 amoebae from a 24-hour culture in Cleveland-Collier medium with addition of rice starch, injected into the liver following laparotomy. To prevent death from bacterial infection, the experimental hamsters were immunized 6 and 3 days before the experiment by intraperitoneal injection of 0·05 ml. of a diluted culture of the bacterial flora. Hepatitis was successfully produced in 95 per cent. of 1,000 hamsters inoculated in the course of 17 months. The inoculation typically gave rise to a single lesion, with occasional metastasis to neighbouring lobes of the liver ; then the lesion rapidly developed until it involved from 25 to 50 per cent. of the liver, with fatal results between 6 and 8 days after infection. On the other hand, injections into the portal vein gave rise to numerous small lesions, showing that trauma of the hepatic parenchyma was not essential. Microscopic examination of stained sections revealed extensive necrosis of the liver parenchyma. The common occurrence of numerous discrete amoebic lesions in the lungs pointed to blood dispersal of the amoebae from the infected liver.

Hepatitis was produced primarily by the amoebae rather than by the bacteria, as shown by the following observations : (1) when bacteria alone were injected, they failed to produce any lesions ; (2) typical amoebic hepatitis was produced in animals treated with a mixture of penicillin and streptomycin and infected with an inoculum of amoebae+bacteria previously treated with these antibiotics ; (3) the lesions were similar both in hamsters which were immunized and those not immunized against the bacteria ; (4) the hepatic lesions were not purulent. Nevertheless, bacteria persisted in the lesions ; therefore they cannot be entirely exonerated until lesions can be produced with bacteria-free amoebae.

It is concluded that hamsters are suitable experimental hosts for the study of extra-intestinal amoebiasis and for testing drugs against amoebic hepatitis. Preliminary trials indicate that emetine and chloroquine, as well as quinacrine [mepacrine] and camoquin, are effective in this condition in hamsters. The lesions are illustrated in four photographs. *C. A. Hoare*

SHOOKHOFF, H. B. *Balantidium coli* **Infection with special reference to Treatment.** *Amer. J. Trop. Med.* 1951, July, v. 31, No. 4, 442–7.

Balantidium coli infection occurs not infrequently in Porto Rico. An incidence of 0·2 per cent. has been recorded, but this study shows a considerably higher incidence, approximately 0·6 per cent. This may be due to the large proportion of children, as no cases were revealed among approximately 1,000 adults, including 16 household contacts of known cases. This suggests that the infection is usually acquired in childhood and has a tendency to die out spontaneously.

This predilection of *Balantidium coli* for children is probably due to their relatively insanitary habits ; thus the incidence of *Ascaris* in those infected was 50 per cent.

From the results of treatment the author considers that diodoquin is to be preferred to carbarsone. The following dosage of diodoquin was employed—

for adults : 0·63 gm. three times daily for 21 days ; for children 1–4 years of age 0·21 gm. three times daily for the same period ; 5–9 years 0·42 gm. t.i.d. for 21 days ; and from 10 years and over the adult dose. In 18 of the 20 cases, there was a history of close contact with pigs. *Philip Manson-Bahr*

NEGHME, A., AGOSIN, M., CHRISTEN, R. & RUBIO, M. Contribución a la quimioterapia del *Balantidium coli*. Comunicación preliminar. [**Chemotherapy of** *Balantidium coli* **Infection : Preliminary Note.**] *Biologica.* Santiago. 1950, July-Dec., Nos. 12/13, 109–16.

The English summary appended to the paper is as follows :—

" The authors demonstrate that Aureomycin and Terramycin exercise a clearly unfavorable action upon the vitality and multiplication rate of *Balantidium coli* ' in vitro '. Minimal active doses are 0·4 mgrs. per cc. of culture medium for the former, and 0·2 mgrs. for the latter ; these doses will produce a clearly marked inhibition of the parasite, while in higher concentrations it will utterly disappear.

" Both drugs seem to act directly upon the Ciliate, as the associated microbic flora suffered only very slight modifications, which showed no direct relation to the doses of Aureomycin or of Terramycin employed. pH does not seem to play any rôle either, as it remained constantly upon an optimal level for the development of the parasite.

" Finally, controls carried out by the authors showed that both drugs seem to act ' per se ' and not due to synergism with Penicillin-Streptomycin, which drugs had been added previously to the culture mediae in order to inhibit the associated microbic flora." [See this *Bulletin*, 1951, v. 48, 156, 471.]

RELAPSING FEVER AND OTHER SPIROCHAETOSES

GEIGY, R. & BURGDORFER, W. Unterschiedliches Verhalten verschiedener Stämme von *Spirochaeta duttoni* in der weissen Maus. [**The Different Behaviour of various Strains of** *Spirochaeta duttoni* **in the White Mouse.**] *Acta Tropica.* Basle. 1951, v. 8, No. 2, 151–4, 3 figs.

Specimens of *Ornithodorus moubata* collected from various localities in Tanganyika and Kenya were examined for spirochaetes at the Swiss Tropical Institute, Basle. As a result six strains of *Sp. duttoni* were isolated and studied in white mice.

Four of these strains were easily established in mice and could be maintained by passage. Another strain was more difficult and after the third passage spirochaetes no longer appeared in the peripheral blood. In the case of a sixth strain, mice could only be infected with very great difficulty and even in the first passage, spirochaetes were not present in the circulation.

A mouse inoculated with a Nairobi strain, one of the first group, showed spirochaetes in the blood up to the 22nd day and then was negative until the 60th day when it was reinoculated with the same strain. The blood was still negative on the 70th day when the mouse was inoculated with a different strain from Ifakara, and after 2 days' incubation period showed a typical infection.

These results confirm the generally accepted view that strains of *Sp. duttoni* from various localities differ considerably in their virulence and immunological behaviour. *E. Hindle*

BLANC, G. & BRUNEAU, J. *Ornithodorus erraticus* **et** *Bufo*. [*Ornithodorus erraticus* **and** *Bufo*.] *Bull. Soc. Path. Exot.* 1951, v. 44, Nos. 5/6, 313–16.

The small variety of *O. erraticus* occurs in abandoned burrows of gerbils (*mérions*) at Saada, near Marrakesh, French Morocco. Probable hosts were lizards (*Gongylus*) and toads (*Bufo mauritanicus*). At Marrakesh, the large variety of this tick occurs in burrows still used by gerbils. The large variety is infected with *Sp. hispanica* but the small variety taken in the abandoned burrows was not found infected in nature.

Experiments with the large and the small variety of *O. erraticus* show that they feed readily on *Bufo* ; a strain is now maintained entirely on toad blood (*Bufo mauritanicus* and *B. viridus*).

Experiments with the large variety of tick naturally infected with *Sp. hispanica* and with the small variety infected with *Sp. merionesi* from laboratory white rats show that the spirochaetal infection in the ticks is not eliminated when a blood meal is taken from *Bufo*. Larval ticks hatched from eggs laid by infected females recently engorged on toads are infective. No spirochaetes were found in the blood of the toads used as hosts for infected ticks. The blood, brain, and liver of toads injected with heavily infected rat blood or guineapig blood remained avirulent. *D. S. Bertram*

LONGANECKER, D. S. **Laboratory and Field Studies on the Biology of the Relapsing Fever Tick Vector** (*Ornithodoros hermsi* **Wheeler**) **in the High Mountains of California.** *Amer. J. Trop. Med.* 1951, May, v. 31, No. 3, 373–80.

A large colony of *Ornithodorus hermsi* was established in the laboratory from 2,747 ticks collected from rodents' nests in the standing remains of dead trees in the areas of Lake Tahoe, and the Big Bear Lake, San Bernardino County, California, at elevations of 6,000 to 7,000 feet. The ticks were kept at a room temperature of 68°F. and a relative humidity of about 90 per cent. and oviposition and development occurred from March to September. Ten out of 26 lots of ticks from Lake Tahoe, and eight out of 13 lots from Big Bear were found to be infected with *Spirochaeta hermsi*, and produced relapsing fever when fed on white mice.

The frequency of transmission of the infection to the offspring of infected ticks was found to be at least 5·3 per cent.

In nature this tick has been found in close association with various rodents and birds including chipmunks, deer mice, bush-tailed wood rats, desert wood rats, bats and mountain blue-birds. It is suggested that dead trees near human habitations should be burnt during the wet season as this would destroy to a large extent the populations of small rodents together with the ticks and fleas associated with them. *E. Hindle*

GILGES, W. **Relapsing Fever simulating Poliomyelitis. Report of a Case.** *South African Med. J.* 1951, Sept. 1, v. 25, No. 35, 622.

The author, from Fort Jameson, Northern Rhodesia, describes a case of relapsing fever in a middle-aged African, who developed a flaccid paralysis of both legs and weakness of the right arm on the fourth day of the first attack. Patellar and Achilles reflexes were absent and Babinski's sign was negative. There was no marked reduction of sensitivity to pain and touch. Laboratory examination revealed little of note, other than the presence of *Trep. duttoni* in the blood. The temperature fell to normal within a week of the beginning of the attack, but a relapse of three days' duration occurred a week later. A similar relapse occurred after 3 weeks.

The patient improved rapidly [treatment is not mentioned]. Within 3 weeks, the left leg could be moved and normal strength had returned to the right arm. The reflexes were now weakly present and the patient could walk unaided within 5 weeks of the start of his illness.

Reference is made to an African girl seen later, brought to hospital with a diagnosis of poliomyelitis, who also recovered rapidly. The diagnosis could not be established but "there were good reasons for suspecting relapsing fever to be the cause of the palsy" which had affected her arms. During the last 15 months, the author saw 132 cases of relapsing fever, most of them "two-day illnesses requiring only symptomatic treatment", but in Indian patients there were many relapses. One African had a facial palsy still present after six months and two children died of acute meningitis: *Trep. duttoni* was found in blood, liver and spleen at autopsy. In a brief reference to the neurological complications of relapsing fever, the author refers to the findings of SCOTT in Libya [this *Bulletin*, 1945, v. 42, 42]. *H. J. O'D. Burke-Gaffney*

HIMPE, N. E. & PIERQUIN, L. Essais de lutte contre *Ornithodorus moubata* au moyen de Cyclotox Solvay. [**Trials of Cyclotox Solvay against** *Ornithodorus moubata*.] *Ann. Soc. Belge de Méd. Trop.* 1951, Apr. 30, v. 31, No. 2, 153–7.

A study was made in the Belgian Congo of the effect on *Ornithodorus moubata* of "Cyclotox", a product of the Société Solvay Co. containing a large proportion of the gamma isomer of benzene hexachloride.

Ticks immersed for one second in aqueous solutions ranging from 0·5 to 50 per cent. all died, those treated with 0·5 per cent. after 80 hours and those at 5 per cent. after 24 hours. When ticks were allowed contact for 1 minute with sand soaked in the same concentration range, those exposed to 5 per cent. and over died, while those exposed to 1 per cent. and under remained alive. Similar tests with sand soaked in 0·5 per cent. and 1 per cent. concentrations showed that, in closed tubes, ticks were killed after 10 minutes' contact with the latter and 30 minutes' contact with the former, when all were dead after 6 hours. In open tubes 0·5 per cent. killed all ticks only after 2 days.

Tests were made in 51 badly infested huts, the sandy floors and bases of the walls being sprayed with 100 cc. of a 5 per cent. aqueous solution to a square metre, giving a final concentration of 0·5 per cent.; 17 huts acted as controls. In sprayed huts, 50 per cent. of ticks had died after 8 days, the survivors being chiefly the larger females. After 10 days all huts were sprayed with a double concentration still giving a 50 per cent. kill in all. The reasons for this partial success compared with the laboratory experiments were that the ticks were not in contact as long, owing to the insecticide being sprayed on, and not mixed with the sand, and that, being a volatile product, its efficacy is soon lost.

Ruth Nash

ERRATUM

In the abstract of a paper relating to the epidemiology of relapsing fever in Somaliland, published in the September 1951 issue of this *Bulletin*, p. 810, and stated to be by EGIDIO, L., the author's name should read LIPPARONI, E.

YAWS

ANGULO, J. J., WATSON, J. H. L., WEDDERBURN, C. C., LEÓN-BLANCO, F. & VARELA, G. **Electronmicrography of Treponemas from Cases of Yaws, Pinta, and the so-called Cuban Form of Pinta.** *Amer. J. Trop. Med.* 1951, July, v. 31, No. 4, 458–78, 24 figs. on 6 pls. [22 refs.]

This paper is based upon the study at the Edsel B. Ford Institute for Medical Research, Detroit, of 30 treponemata from a Jamaican case of yaws (a Negress aged 2 years), 9 treponemata from a Mexican case of pinta (an adult Indian) and 89 from a Cuban case of pinta (an elderly Negro in whom the disease had been studied for 20 years and in whose lesions treponemata were always plentiful [this must be an unusual occurrence in the other treponematoses]). All cases were clinically typical and untreated. The preparations tended to be electronopaque so they were shadowcast with chromium at an angle of about 12°.

The mean length and width of 10 chromium-shadowed *Treponema pertenue* were 11·4 (8·16–15·1) and 0·13 (0·08–0·21) microns and those for the 33 chromium-shadowed treponemata from the case of Cuban pinta measured $14{\cdot}03 \pm 4{\cdot}78 \times 0{\cdot}15 \pm 0{\cdot}027$ microns. It is claimed that these are the first measurements of these organisms based upon electron micrographs.

The appearances of the three treponemata are discussed and " no differences in morphology or structure were detected among these species or with regard to *T. pallidum*, except for the lack of flagella shown by *T. pertenue* specimens which was probably an artifact ".

[This study needs extension and perhaps treponemata from the same stage of infection should be studied, that is, either from animal infection or from human volunteer infections of the same duration.] *C. J. Hackett*

HILL, K. R., KODIJAT, R. & SARDADI, M. **Atlas of Framboesia. A Nomenclature and Clinical Study of the Skin Lesions.** *Bull. World Health Organization.* Geneva. 1951, v. 4, No. 2, 201–46, 42 figs. on 32 pls. [12 refs.] Reprinted as *World Health Organization : Monograph Series*, No. 5, 1951, 52 pp. [Price 5*s*.]

The authors state that " the reason for the communication is that we feel that the present diverse and confusing descriptions of framboesia should be clarified and, as far as possible, accurate descriptions given ". In the next paragraph they say " no attempt has been made here to study the histopathology of framboesia ; the descriptions given relate to what is seen by the naked eye ". [This restriction must limit the outlook of the authors and the value of the publication. No reference is made to South American work and only one paper from French workers is referred to.]

The initial lesion is called the initial framboesioma, and apart from an initial framboesial ulcer and gangosa, all other skin lesions are varieties of framboesides. No account is taken of secondary or tertiary stages because " in our experience certain framboesides usually appear early and others usually late ; but there is no certainty about this, and we have studied cases in which nearly all the varieties of framboesides have commenced their courses within a year or so, many of them coincident with each other ".

[The references to granulation tissue in the fungating framboesioma, and to ulcerative plantar framboesides (granulomatous plantar yaws), stress the need for pathological control. This is also needed when primary lesions " apparently change their nature to resemble late ulcerative framboesides ". The reviewer would not agree to equate rupial lesions of secondary yaws with the authors' rather confused erythematous framboeside. The whole interesting

problem of the hand and foot lesions apart from granulomatous yaws in these regions is got over by referring to them as non-ulcerative palmar and plantar framboesides and saying that they are protean in appearance. Some of the 42 illustrations are not in sharp focus and often both soles are shown when a larger picture of only one would have been more useful. The reviewer agrees that there is need for an internationally acceptable nomenclature of yaws lesions, but this must be based upon the pathological changes causing them and all aspects of the course of the disease.] *C. J. Hackett*

PAYNE, E. H., BELLERIVE, A. & JEAN, L. **Chloromycetin as a Treatment for Yaws and Tropical Ulcer.** *Antibiotics & Chemotherapy.* New York. 1951, Apr., v. 1, No. 1, 88–91.

The first trials of chloromycetin [chloramphenicol] by mouth in the treatment of secondary yaws in Haiti were started in December 1949. The dose was either 1 gm. repeated every half-hour for 3 doses (total 3 gm.) or an initial dose of 2 gm. followed by 0·25 gm. every 3 hours to a total of 16 gm. Patients of another series were given 2 gm. intravenously each morning for 5–6 days (total 10–12 gm.). The only results reported are that at the end of the second week after treatment had started, secondary lesions were completely healed and encrusted and that primary lesions healed more slowly (1–3 weeks). Pain had gone from all yaws at the end of a week. [No serological observations after treatment are recorded.] At least 21 of the yaws' patients treated with chloramphenicol also had tropical ulcers. Within 7–10 days new epithelium appeared at the edge of the ulcer and " within 6 weeks many of the lesions showed satisfactory healing ". *C. J. Hackett*

LEPROSY

BADGER, L. F. **History of Leprosy in the United States.** *Ann. New York Acad. Sci.* 1951, Mar. 30, v. 54, Art. 1, 6–11.

The author acknowledges that the number of cases of leprosy in the U.S.A. is not known. The most reliable statistics are those provided by the National Leprosarium in Carville during the 30 years that it has been in existence. During that period patients born in 52 different political areas have been admitted in the following percentages : Mexico 33, Pacific Islands 20, West Indies 13, China 9, Italy and Greece, each 5. During these 30 years 1,500 patients, a yearly average of 50, were admitted from 41 States and the District of Columbia. With the exception of Florida, Louisiana and Texas, which are looked upon as endemic States, the great majority of cases have occurred in foreign-born persons. Cases of leprosy in those native to the State from which they were admitted have entered Carville from only 21 of the remaining 38 States, and the majority of these had served abroad in endemic countries or resided in endemic areas in the U.S.A. Cases have been noted, however, in some of the non-endemic States in persons who have never been outside the State and give no history of contact with a recognized case. During the period there were admitted to Carville 551 patients from 4 States : California, Florida, Texas and Louisiana, these being the States in which they were born.

The laws regarding leprosy vary from State to State. The laws of several States still require that every case of leprosy, regardless of type, should be isolated. Efforts at a control programme have been sporadic and of brief duration for the most part. There are, however, indications that the trend of

the incidence in most regions of the country is downwards. Special stress is laid on the incidence in New York State from which 150 cases have been sent to Carville in the last 30 years.

The author considers that there is no assurance that leprosy will not spread in the future even in the northern States. "Where the factors influencing spread exist new cases will develop, north and south. One must be continually on the outlook for new cases developing among contacts with recognised and unrecognised cases." *Ernest Muir*

DE LA LASTRA SOUBRIER, J. M. Epidemiologia de la lepra. [**Epidemiology of Leprosy.**] *Med. Colonial.* Madrid. 1951, June 1, v. 17, No. 6, 567–87.

This is an excellent review of the previous work on this subject, but it contains no original contribution of work by the author. *Ernest Muir*

ROMERO L., A. I. Contagio familiar y extrafamiliar en lepra. II. Estudio de los convivientes que se encontraron enfermos de lepra en el periodo que va de 1946 a Agosto de 1950. [**I. Contagion inside and outside the Family. II. A Study of Contacts of Leprosy Patients in the Period from 1946 to August 1950.**] *Rev. Méd. Costa Rica.* 1950, Nov., v. 9, No. 199, 271–4, 274–77. English summary.

I. Of the 272 patients in the *Sanatorio de las Mercedes* 65·5 per cent. contracted leprosy inside the family, and 34·5 outside the family. Those infected inside the family also had the disease in a worse form. From these two facts the author concludes that prolonged contact is necessary to produce contagion and that there is a familial hereditary predisposition. [It would surely be better to place the blame on the much closer contact inside the household.]

II. Out of 1,084 contacts of leprosy patients, 60 new cases were discovered, 21 of them being lepromatous, 9 undifferentiated but bacteriologically positive, 23 undifferentiated negative, and only 7 tuberculoid. The contacts were as follows: 15 sleeping on the same bed, 17 in the same room, 10 in the same house and 18 through visits to outside houses. *Ernest Muir*

MALFATTI, M. G. Investigaciones sobre la morfologia del *Mycobacterium leprae* a traves de la optica electrónica. [**Investigations on the Morphology of *Mycobacterium leprae* by means of the Electron Microscope.**] *Semana Méd.* 1951, July 19, v. 99, No. 3, 109–18, 10 figs. English summary.

This article is illustrated with 9 photomicrographs with a magnification of 3,500 diameters, and with a drawing. The lepra bacillus is seen to consist of a dense rod surrounded by a periphery of clear material. In the cytoplasmic rod there are granules which are sometimes equally distributed, but more often gathered in concentrations of different sizes at the poles or in the centre. The bacilli vary in length from 2 to 6 microns, and in breadth from 0·3 to 0·5 micron. Some are seen in division through the middle of their length. The bacilli were taken from an untreated L2 case, and it is the intention of the author to compare these with the appearances in treated cases. The material was taken from a superficial puncture of the skin without any blood, diluted with distilled water and dried at 37°C. It is hoped to be possible to distinguish *Myco. leprae* from other mycobacteria by their different appearances under the electron microscope. *Ernest Muir*

FELDMAN, W. H., KARLSON, A. G. & GRINDLAY, J. H. **Lepromin : Mitsuda's Reaction with Experimental Observations in Dogs.** *Ann. New York Acad. Sci.* 1951, Mar. 30, v. 54, Art. 1, 53–72, 8 figs. [36 refs.]

This paper consists of two parts. In the first, types of antigen, characteristics of the reaction, value of the test, and certain theoretical features are discussed. The possibility is suggested of the "hypersensitization of previously non-sensitive individuals as a consequence of the gradual liberation of soluble, antigenic fractions from the bacilli contained in the lepromin. As a consequence the mechanism of reaction is stimulated sufficiently to incite in some individuals a focal tissue reaction at the site where the antigen was introduced". In this way a positive reaction could occur in those who had had no contact with *Myco. leprae*. [No suggestion is offered as to why the mechanism of reaction should be interfered with in lepromatous cases.] The following statement is made : "it is generally accepted by leprologists that failure to react positively to lepromin implies susceptibility to infection with *Myco. leprae* and that a positive reaction indicates protective resistance. [This is very questionable ; most leprologists take the view that only in those definitely diagnosed on clinical or bacteriological grounds as suffering from leprosy can we be sure that the lepromin test indicates the degree of resistance. We know of no evidence that there is any reliable indication in those not thus diagnosed.] The second half of the paper recounts a study of the lepromin test in dogs. Wade's experiments in a region where there was much leprosy are confirmed by similar experiments in a region where no leprosy was known to occur. The data obtained showed that dogs vary in their capacity to develop hypersensitivity to lepromin ; that the initial injection of lepromin often increases this hypersensitivity and causes increased tissue response ; the latter implies the elaboration and influence of circulating activating substances ; the reaction of dogs to lepromin is not necessarily dependent on previous contact with *Myco. leprae ;* lepromin injection in dogs did not sensitize them to a diagnostic dose of Old Tuberculin.

Ernest Muir

USTVEDT, H. J. **Observations on the Lepromin Test.** *J. Oslo City Hosps.* 1951, May, v. 1, No. 5, 117–21.

Following on the theory of CHAUSSINAND and others that it may be possible to protect human beings against leprosy by means of BCG, experiments were made with Dharmendra's type of lepromin antigen. Inoculation of 17 patients with active tuberculosis showed no correlation between sensitivity to tuberculin and sensitivity to lepromin. In 5 persons without tuberculous disease, but with positive tuberculin reactions the lepromin test was positive in 4, while in 7 non-reactors to tuberculin reactions to lepromin were all negative. Of 4 BCG-vaccinated reactors to the adrenaline-Pirquet test (A.P.) 3 were reactors to lepromin ; but of 12 who were negative to AP but positive to Mantoux, only one was positive to lepromin. Out of 11 with negative Mantoux before vaccination with BCG, 3 converted to positive AP reaction and positive lepromin after BCG vaccination. Seven converted to positive Mantoux, but none of these converted to a positive lepromin. Thus those who are positive to Mantoux T.U.100 (but negative to adrenaline-Pirquet) after vaccination with BCG have only in rare cases shown definite positive reactions to lepromin, but reactors to AP are often, but not always, reactors to lepromin.

Ernest Muir

ROSEMBERG, J., SOUZA CAMPOS, N. & AUN, J. N. Da relação imunobiológica entre tuberculose e lepra. IV. A lepromino-reação em crianças vacinadas um ano antes com BCG, descendentes de doentes de lepra. Dissociação entre alergia tuberculínica e reação de Mitsuda. [**Immunobiological Relation between Tuberculosis and Leprosy. IV. The Lepromin Reaction in Healthy Children of Leprous Parents, BCG vaccinated a Year before the Test. Dissociation between Tuberculin Allergy and Mitsuda Reaction.**] *Rev. Brasileira Leprologia.* S. Paulo. 1951, Mar., v. 19, No. 1, 8–18. English summary.

Thirty-eight healthy children of leprous parents aged 1 to 18 months, who had been isolated at birth, were inoculated with Mitsuda antigen and at the same time given BCG by mouth. Of the thirty-eight, 12 received a single dose of 0·10 gm. BCG and the other 26 received progressive daily doses for 28 days with a total of 1·19 gm. Of the 12 who had a single dose of BCG, 4 had a negative lepromin reading, but the other 8, along with the 26 who had progressive doses of BCG, all became lepromin-positive. A year later all the children had the lepromin test repeated, and this time all were positive including the 4 that had been negative before. It was observed however that there was no relationship between the tuberculin sensitiveness and the lepromin reaction since two cases were never found positive to Mantoux (1 : 10), and of the 36 others who developed allergy 29 became entirely negative to the Mantoux test 6 to 11 months before the second lepromin test to which 100 per cent. were found positive. *Ernest Muir*

ROSEMBERG, J., SOUZA CAMPOS, N. & AUN, J. N. Da relação imunobiológica entre tuberculose e lepra. V. Tempo de positivação da reação de Mitsuda após a introdução simultânea de BCG por via oral e de lepromina por via intradérmica. [**Immunobiological Relation between Tuberculosis and Leprosy. V. Time of Positivation of Mitsuda's Reaction after the Simultaneous Introduction of BCG (Orally) and Lepromin (Intradermally).**] *Rev. Brasileira Leprologia.* S. Paulo. 1951, Mar., v. 19, No. 1, 19–26. English summary.

This paper is next in series with the last, the findings of which it confirms and amplifies. Forty-five healthy children of leprous parents, aged 5 days to 11 months, who had been isolated at birth, were found negative to the Mantoux test (1 : 10). They were tested with lepromin, and at the same time oral administration of BCG was begun. In 30 of them, 0·10 gm. of BCG was given once a week for three weeks ; the other 15 children were kept as controls. The lepromin test was entirely negative in the 15 controls, but positive in every one of the 30 who had BCG. The development of the positive reaction appeared at different intervals after inoculation : 3 after 23 days, 8 after 30 days, 15 after 40 days, 3 after 50 days, and 1 after 60 days. In 7 cases there was an erythematous and infiltrative reaction round the point of inoculation after 48 hours, and in 2 others the same occurred on the 9th day, *i.e.*, 48 hours after the second ingestion of BCG. The authors consider it an established point to look upon the positive lepromin reaction as a proof of resistance to *Myco. leprae*, and that it would be interesting to consider this type of vaccination on a mass scale, especially in new-born children of leprous parents who could not be promptly isolated. *Ernest Muir*

PINTO, J. T. & ZEO, A. **Universal Serologic Reaction in Leprosy.** *Ann. New York Acad. Sci.* 1951, Mar. 30, v. 54, Art. 1, 48–52, 3 figs.

Using KAHN's Universal Serological Reactions [this *Bulletin*, 1949, v. 46, 944 ; 1951, v. 48, 997] the authors tested a group of 75 patients with leprosy of

different types and with varying degrees of activity. They confirmed the characteristic leprosy pattern described by Kahn in some cases only ; in others it was absent. In some the pattern became apparent after promin treatment. The degree of precipitation was directly related to the degree of activity of the disease, but in very active cases precipitation was low, presumably because of the patient's inability to form antigens. It has been claimed that the pattern given by lepromatous leprosy is distinct from that of syphilis, yaws, malaria and tuberculosis. *Ernest Muir*

BOENJAMIN, R. **Vitamin A and Carotenoids in the Blood Serum of Lepers and of their Healthy House-Mates.** *Documenta Neerlandica et Indonesica de Morbis Tropicis.* Amsterdam. 1951, June, v. 3, No. 2, 147–54.

The contents of vitamin A and carotenoids in the blood serum of patients with lepromatous leprosy was somewhat lower than in normal persons. In patients suffering from the neural type this content was normal. There was no indication that a decreased intake of vitamin A enhanced the susceptibility to leprosy. *R. Passmore*

REISS, F. **The Detection of Leprosy by the Dermatologist.** *Ann. New York Acad. Sci.* 1951, Mar. 30, v. 54, Art. 1, 73–83, 19 figs.

This is a clearly written and well illustrated description of the clinical signs of leprosy in its different types and stages, which should be useful to medical practitioners who see only occasional cases of the disease. *Ernest Muir*

ELLIOTT, D. C. **An Interpretation of the Ocular Manifestations of Leprosy.** *Ann. New York Acad. Sci.* 1951, Mar. 30, v. 54, Art. 1, 84–100, 4 figs. [36 refs.]

The use of the slit lamp has made possible much earlier recognition of ocular lesions in leprosy, and shows that these occur much earlier in the course of the disease and in a much larger proportion of cases than was formerly supposed. The author reviews the recent work of a number of those who have studied eye leprosy and also writes from his own study of eyes of a thousand leprous patients. Among the interesting conditions described are the following : the sleeve-like nodules along the course of the corneal nerves, especially at the branching points of the finest branches ; the fixation of Hansen's bacillus in capillary tufts at the limbus ; pigment-studded fibrin strands in the anterior chamber, denoting early iris infection. A particularly interesting case of leprosy in a child of four is described. The child, the daughter of a leprous mother, had only one skin lesion so slight that it would have passed unnoticed under other circumstances. "After repeated examinations of this child, we were able clearly to demonstrate the perineural beading infiltration along the course of the corneal nerves, together with early destructive changes on the anterior surface of each iris. There were wisps and floating leucocytes with strands of fibrin imbedded with particles of pigment drifting in the anterior chamber."

The author, who is not only an ophthalmologist but the chief medical officer of the Veterans' Administration Office, stresses the importance of eye examinations in the early diagnosis of leprosy, especially in veterans from the last World War and those now returning from Korea, where there are as many as 35,000 open cases of leprosy in the southern part. After the Spanish-American war it took 10 to 25 years after exposure before cases were diagnosed ; skill in eye examination may speed up diagnosis this time.

The reason given for initial lesions being at the limbus is that it is there that there is active fusion of the superficial and deep vessels. This article is full of interesting and valuable information, and should be read in the original.

Ernest Muir

ROMERO L., A., CASTRO JENKINS, A. & ALVARADO, R. Complicaciones renales en los enfermos de lepra. [**Renal Complications in Leprosy Patients.**] *Rev. Méd. Costa Rica.* 1950, Nov., v. 9, No. 199, 244–9. English summary (9 lines).

PINEDA is quoted as saying that 16 per cent. of leprosy patients die of nephritis and RAKE found renal lesions in 29 per cent. of autopsies. The authors during the period from 1945, when they began sulphone treatment, up till the end of 1949 examined the urine every month of 214 patients treated with sulphones. Of these, 16 died during the 4 years, and in only 1 of them could death be attributed on clinical grounds to glomerulonephritis. Studying the results of urine examinations the authors found that during lepra reaction urine often contained tube-casts, blood and traces of albumin, which, however, disappeared a few days after the reaction cleared up. There were 9 cases with constant albuminuria during the 4 or 5 years, and these showed occasional casts and red cells. There were also 3 cases with occasional albuminuria. In these 12 cases the authors are of the opinion that the renal condition originated in severe and prolonged attacks of lepra reaction. Sulphone treatment was continued in all these cases without any effect, good or bad, on the urinary syndrome. They consider that the toxins set free from lepra bacilli during reactions cause parenchymatous lesions of the kidneys.

Ernest Muir

ERICKSON, P. T. **The Treatment of Leprosy.** *Ann. New York Acad. Sci.* 1951, Mar. 30, v. 54, Art. 1, 115–25. [19 refs.]

In this review the former treatment with chaulmoogra oil and its derivatives is compared with the more recent forms of treatment, especially the sulphones. At Carville the chances of recovery after different periods of treatment rise from 3·6 per cent. of arrests by the end of the second year, to 28·7 per cent. by the end of the fifth year, and 73 per cent. by the end of the ninth year.

Much of the contents of this paper was already reported in a former paper [this *Bulletin*, 1951, v. 48, 1001]. Limited experience with cortisone has shown that it might be of value in the control of lepra reactions. In the discussion which followed the reading of this paper, Dr. Marcel ROCHE told of the administration of ACTH for periods up to one week to six patients with lepromatous leprosy who were undergoing a lepra reaction. "In all the cases there was a striking and rapid partial remission in most of the characteristic symptoms of the reaction throughout the period of ACTH administration. The enormous increase in appetite and the feeling of well-being as well as the objective evidences of remission were felt..... to be quite remarkable."

Ernest Muir

LAVIRON, P. & LAURET, L. Les injections intraveineuses d'huile de chaulmoogra dans le traitement de la lèpre. [**The Intravenous Injection of Chaulmoogra Oil in the Treatment of Leprosy.**] *Rev. Coloniale de Méd. et Chir.* 1950, Jan. 15, v. 22, No. 174, 2–6. [12 refs.]

Pure neutralized chaulmoogra oil had been injected intravenously in doses of 2 cc. twice a week since 1936 with good results. As the result of the recommendation at the Havana Congress to use larger doses of chaulmoogra, the

twice weekly amount was raised to 5 cc. thrice a week, with a yearly total of 525 cc. in place of 150 cc. With this increased dosage the percentage and degree of amelioration were very considerably increased, as is shown in two tables in the text. The oil used is that of *Hydnocarpus wightiana*. Its acidity is usually 4 to 6 per cent. and it is neutralized by adding 1,200 gm. of powdered sodium carbonate to 30 kgm. of oil and stirring for one day. This forms soap of the fatty acid, and at the end of the operation the acidity is reduced to 0·08 to 0·12 per cent. After filtering, this neutralized oil is well tolerated intravenously in these large doses, and does not cause sclerosis of the veins. The only bad effect, which is very occasional, is a feeling of depression, and this is dispelled by lowering the dosage. In 1948-49 only two out of 63 cases had to change the treatment because of generalized pains and fugitive swelling in the limbs. *Ernest Muir*

LAVIRON, P., LAURET, L. & SCHNEIDER, J. Traitement de la lèpre par des injections espacées de D.D.S. dans le chaulmoograte d'ethyle. [**Treatment of Leprosy with Injections at long Intervals of DDS in Ethyl Esters of Chaulmoogra.**] *Bull. Soc. Path. Exot.* 1951, v. 44, Nos. 5/6, 285–9, 8 figs. on 4 pls.

This is an account of 67 patients treated in W. Africa for periods of 3 months to a year with a suspension of DDS in the ethyl esters of chaulmoogra oil. A 25 per cent. suspension was made up in glass capsules of 5 cc., and this amount was injected into the muscles every 15 days, with a reduced dose for children. The patients complained of local pain for 24 to 48 hours, but after a year's treatment there was no sign of nodules or induration at the points of injection. Of 35 of those who had been treated for 6 months or more there were 2 stationary, one was intolerant and had to stop treatment, 15 had +++ improvement, 13 had ++ improvement and 3 had + improvement (one is not accounted for). There was lepra reaction in 7. The patients stated that they preferred this form of treatment to taking daily DDS tablets. The results were just as good as with the latter form of treatment. It was found advisable to give a month's rest from treatment after every 3 months' period of treatment. The results are illustrated in 4 pairs of photographs. *Ernest Muir*

LOWE, J. & DAVEY, T. F. **Four Years' Experience of Sulphone Treatment of Leprosy.** *Trans. Roy. Soc. Trop. Med. & Hyg.* 1951, June, v. 44, No. 6, 635–62. [18 refs.]

This article repeats much of the information in a previous article by LOWE [this *Bulletin* 1951, v. 48, 1000]. There are, however, some new matters of interest. The results of sulphone treatment in the tuberculoid type of leprosy in 50 cases are "considered highly satisfactory, being produced with a high degree of certainty and within a reasonable time. One year to 18 months appears adequate in these cases, although relapse has been seen in one case treated for 12 months." The possibility of streptomycin in controlling complications and speeding up response to treatment is mentioned, preliminary experiments having shown its activity in leprosy, but its high cost will limit its use greatly in countries such as Nigeria. It is particularly useful in tuberculoid cases. There are 20,000 cases on DDS treatment in Nigeria in many widely scattered centres. The treatment is carried on by African assistants with occasional visits by a doctor. Thiacetazone [Conteben, TB.1/698] administered alone is giving promising results in all types of cases, but an attempt to give it in combination with DDS is meeting with difficulties.

Under *Pharmacology of Sulphones* the following notes are of interest: On the usual oral doses, sulphetrone 3 to 6 gm. a day and diazone 0·9 to 1·8 gm. a day, the patient's blood shows the presence of free DDS 0·2 to 0·6 mgm. per cent., which is a therapeutic blood level, and is equal to that produced by giving DDS itself, 200 mgm. a day or 300 to 400 mgm. twice a week. That the complex sulphones are hydrolysed to DDS mainly in the stomach is shown by the finding that injected sulphones produce very low levels of DDS in the blood. "A new finding of some interest is that DADPS [DDS] is not excreted in the urine unchanged, as others have recorded, but is mainly in the form of a water-soluble metabolite. This probably explains the complete absence of any urinary complications." *Ernest Muir*

CONNOLLY, Evelyn M. **Some Notes on the Sulphetrone Treatment of Leprosy.** *East African Med. J.* 1951, July, v. 28, No. 7, 278–9, 8 figs. on 4 pls.

Forty-five lepromatous cases in a Mission station in Uganda were treated with sulphetrone tablets for 2 years. Nodules were cleared up entirely in 44·4 per cent. and lesions diminished in 46·6 per cent. Bacteriologically negative findings were obtained in 46·4 per cent., and bacilli were fragmented or greatly diminished in number in 44·4 per cent. The paper is illustrated by 7 photographs and one photomicrograph. *Ernest Muir*

WHEATE, H. W. **Preliminary Report on Sulphetrone Therapy in Lepromatous Children.** *East African Med. J.* 1951, July, v. 28, No. 7, 272–7, 8 pls.

Twenty-five children in a Leprosy Colony in Uganda were treated with sulphetrone tablets for periods varying from 6 to 20 months, in doses rising from 0·5 gm. to 3 gm. per day during 6 weeks, after which a week's rest was given. The haemoglobin test was taken, and iron and yeast administered, the latter being discontinued later with no ill effects. There was marked clinical improvement in 12 cases and moderate in 11. One case became bacteriologically negative and 17 showed moderate bacteriological improvement. There was lepra reaction in several cases, followed by improvement. The paper is illustrated by 8 photographs of 4 patients. *Ernest Muir*

MEREDITH, J. S. & PATTON, G. Q. **A Preliminary Report on the Use of D.A.D.P.S. in Leprosy.** *East African Med. J.* 1951, May, v. 28, No. 5, 197–203.

This is an account of the treatment of 37 lepromatous and 123 tuberculoid cases of leprosy with DDS for a period of 6 months, in the north-east corner of Northern Rhodesia. Reading LOWE's article on DDS treatment in Nigeria [this *Bulletin*, 1950, v. 47, 475] the authors judged that it might be possible to introduce this treatment in spite of the absence of a fully equipped laboratory. The original schedule proposed by Lowe was found excessive, but in one of the two treatment centres a daily dose of 300 mgm. was reached, and in the other 200 mgm. Three types of reaction were produced: a febrile exacerbation of lesions similar to ordinary lepra reaction; exfoliative dermatitis, which required the cessation of treatment for two months; oedema and joint pains, in which it was found sufficient to reduce the daily dose to 100 mgm. No evidence was seen "of the type of reaction described by Lowe in which there is a condition similar to infectious mononucleosis". Treatment was given by mouth because of the ease of administering tablets. In one of the two groups there were no deaths, but in the other there were nine, four being from pneumonia. It was considered, however, that there was no connexion between these and

the DDS treatment, as they occurred in the old-age group, were at the coldest time of the year and none of the patients suffered from any form of reaction at the time. The authors consider a trial of six months too short to evaluate the treatment, but they report that " within a very short time, usually about two months from the start of treatment, there is a noticeable improvement in (1) the mental attitude of the patients, (2) the general physical condition ". This is in spite of the obvious inadequacy of diet : " owing to food shortages we have had to fall back on cassava as the staple diet in place of millet. As a result the protein intake is less than it was before. In spite of that, there is a steady improvement in the general physical condition of almost all the patients even the very old asthenic ones." [This report is of value principally as showing that DDS can be safely administered in tablets, with a minimum of laboratory facilities, to patients on the most inadequate diet, unselected as to their type and general physical condition, and yet with valuable results. All patients received an iron preparation as a routine and in only one was there a striking fall in haemoglobin percentage. It is worthy of note that 100 mgm. a day was found to be a safe but effective dose after initial experimentation with larger amounts.] *Ernest Muir*

ALLDAY, Elizabeth J. & BARNES, J. **Toxic Effects of Diaminodiphenyl-Sulphone in Treatment of Leprosy.** *Lancet.* 1951, Aug. 4, 205–6.

One hundred and fifty-three cases of leprosy were treated for periods varying from 2 to 8 months with DDS. The daily oral dose was 200 mgm. slowly reached within 2 weeks from an initial dosage of 100 mgm. The object of the paper is to relate the toxic effects which were observed in some of the patients. Apart from lepra reaction, psychosis in one case, slight headache which soon passed off, and hypochromic anaemia which did not consitutute a serious problem, the main toxic effect noted in 7 (4·5 per cent.) of the patients was a syndrome with a series of symptoms. The usual history was that the patient had been taking DDS for 5 or 6 weeks " when suddenly, out of the blue, comes a violent illness whose presenting symptom is a papular or exfoliative rash. The papules may be 2 to 7 mm. in diameter. The distribution varies, the rash being either confined to the upper limbs or to the forehead or widely disseminated over the whole skin, only the scalp, palms and soles escaping . . . fever, malaise and weakness are present from the beginning and accompany the rash . . . Within a few days other symptoms of internal disorder follow : enlargement and tenderness of the liver, epigastric and hypochondriac pain, jaundice, swelling of the lymph glands, notably along the posterior margin of the sterno-mastoid muscles, and mononucleosis (lymphocytes and monocytes 70 per cent.) . . . For the next week the disease seems to hang in the balance before inclining to a gradual improvement or death, which took place in one case after 16 days ". LOWE, who had somewhat similar toxic signs in some cases [this *Bulletin*, 1950, v. 47, 475], attributes the glandular enlargement and mononucleosis to the activation of the latent virus of glandular fever, because of a positive Paul-Bunnell reaction ; but the authors of this paper are not convinced of this, as in the one case in which they had a Paul-Bunnell test done it was negative. Because of the toxicity with 200 mgm. per day the dose was reduced to 100 mgm., but there was another case of exfoliative eruption with glandular enlargement, and the patient was gravely ill.

[The fact that the only places where symptoms of this nature have been reported are in the adjoining Ogoja and Owerri Provinces of Nigeria suggests that DDS has activated some latent infection not found, or at least not so common, in the many other parts of the world where it has been used. The 153 patients are reported as being " all particularly strong and healthy adults "

and anaemia was not a serious problem ; so the symptoms can scarcely have been the direct result of DDS. It should also be pointed out that by the latest standards 200 mgm. per day is considered a dangerous dose in advanced lepromatous cases except under very careful daily experienced supervision.]

Ernest Muir

LANGUILLON, J. Les résultats du traitement de 15 cas de maladie de Hansen à forme lépromateuse par le 4-4'-diamino-diphényl-sulfone. [**Treatment of 15 Cases of Lepromatous Leprosy with 4-4'-Diaminodiphenylsulphone.**] *Bull. Soc. Path. Exot.* 1951, v. 44, Nos. 5/6, 322–9.

This account is from Guadeloupe. The 15 patients were given a series of courses of DDS, each lasting for 8 weeks, during which time the daily dose gradually rose from 100 to 200 mgm. There was an eight-day rest between courses. The author reports that after 7 months or less of treatment, 11 cases became bacteriologically negative and of these 7 had no clinical signs left.

Ernest Muir

MONTESTRUC, E. & BLACHÉ, R. Considérations sur les modifications bactériologiques apportées par les sulfones dans la lèpre à type lépromateux. [**Considerations on the Bacteriological Changes caused by the use of Sulphones in the Lepromatous Type of Leprosy.**] *Rev. Coloniale de Méd. et Chir.* 1950, May 15, v. 22, No. 178, 110–12.

Some 209 patients of whom 115 were of the lepromatous type were treated with sulphones (chiefly diasone) in the island of Martinique for two years. The treatment brought about a remarkable diminution in the bacteriological infection, especially in early cases. The authors stress the importance of early and continuous treatment.

Ernest Muir

MUIR, E. **Bacteriological Changes under DDS Treatment of Leprosy.** *Leprosy in India.* 1951, Apr., v. 23, No. 2, 116–26, 1 graph.

This paper is a continuation of one published a year earlier [this *Bulletin* 1951, v. 48, 375]. A rough method is devised for assessing the bacillary index (B.I.) of patients in counts from 0 up to 20. It is thus possible to estimate the bacteriological improvement or otherwise at each three-months' examination. Out of 100 patients on DDS treatment, 58 who had been under treatment for periods of 12 to 18 months were chosen and the effects of treatment analysed. These patients were of the lepromatous type in different stages of the disease. They were examined daily and given the maximum dose that they were considered able to tolerate. In some, on account of weakness, this was a very small amount. In 12 such patients, who were only able to tolerate half the amount tolerated by strong healthy patients, there was an improvement in the B.I. of 50 per cent. At first, patients were given ferrous sulphate as a routine, but later this was found not to be necessary. An analysis of the figures of those suffering from lepra reaction showed that those with reaction lasting more than 100 days of the period had a decrease of the B.I. of 24·3 per cent., whereas those with reaction for less than 10 days had a decrease of only 21·3 per cent., although the former naturally included the most advanced and weakest patients. On the other hand the importance of early treatment is shown by the fact that in patients with a B.I. of 10 or less there was a reduction of the B.I. by 48·5 per cent. In a group of 9 patients who were intolerant of DDS on account of severe reactions and anaemia, it was found that tolerance was attained by injections of vitamin B_{12}. The patients were first able to stand increased doses,

and this was accompanied by an increase in the haemoglobin. It is demonstrated that 50 mgm. of DDS a day or even less is therapeutically active in the adult of average weight. *Ernest Muir*

GARDUÑO, D. M. & DE LEON, D. M. **Streptomycin in Leprosy, Early Cutaneous Type.** *J. Philippine Med. Ass.* 1951, Feb., v. 27, No. 2, 93–101. [10 refs.]

A single case of leprosy treated with 65 grammes of dihydrostreptomycin, spread over a period of 130 days, is described. The patient, a Filipino, was also found to be suffering from malaria and pulmonary tuberculosis. The exact type of leprosy was doubtful as the lesions appeared to be tuberculoid, but bacilli were found, indicating an early lepromatous case, as did also the histological findings. After 13 days of injections there was a flare-up of the skin lesions and new patches appeared ; this subsided on the 43rd day. On the 92nd day bacteriological findings were negative for the first time. On the 117th day there was another flare-up after which the lesions gradually faded, and the patient left hospital six months after beginning treatment, the skin lesions having cleared up. He had gained weight and had only occasional cough.

The lepromin test was apparently not done until 2½ months after the last streptomycin injection. It was negative for the first two weeks, doubtful in the third, single + in the fourth and doubtful in the fifth. The significance of the test is discussed. *Ernest Muir*

ROLLIER, R., NOURY, WEISGERBER & MAURY. Essai de traitement de la lèpre par une variété de Salsepareille. [**Trial of the Treatment of Leprosy with a Variety of Sarsaparilla.**] *Maroc Méd.* 1951, Sept., v. 30, No. 316, 776–80.

The authors were led to this form of treatment by a description by Dr. ARNAUD of the custom of the Arabs of S. Morocco of treating their syphilis cases with "hachba", which is the local name for sarsaparilla. They noticed by chance that a lepromatous patient who had undergone this treatment had got rid of all signs of the disease. Sarsaparilla can be given in the form either of a decoction or of an 8·2 per cent. soft extract. With the former, 15 gm. of the powdered root of *Smilax ornata* is boiled in sufficient water for a quarter to half an hour, and this is taken twice a day for an indefinite period. Of the soft extract 2 to 4 "dragées" of 0·3 gm. are taken morning and evening, according to tolerance, for 6 days a week in courses of 3 months, a month's rest being allowed between courses. Of the 25 patients treated 18 were lepromatous, and of these there was much improvement after 5 or 6 months in 3, and moderate improvement in 10. Only in one was there no improvement. In most cases there was amelioration in nasal conditions. In 7 neural cases there was improvement in the hand deformities. The complications were not severe. In some there was slight anaemia, but this yielded to vitamin B_{12} and vitamin B_6. Gastric irritation was relieved by alkalis. The authors also found bacteriological improvement after 4 months. They found that the forms of improvement were similar to those with sulphones. They have three patients on a combined treatment of sarsaparilla and sulphones, but it is still too soon to report results. They have the impression that sarsaparilla alleviates better than sulphones the anaesthesia and deformity of polyneuritic phenomena. *Ernest Muir*

ROMERO L., A. Uso de la vitamina "E" en las atrofias musculares que presentan los enfermos de lepra. [**The Use of Vitamin E in the Muscular Atrophies found in Leprous Patients.**] *Rev. Méd. Costa Rica.* 1950, Nov., v. 9, No. 199, 260–63. English summary.

Out of the 164 patients at the *Sanatorio de Las Mercedes*, 32 presented deformities and muscular atrophies of the hands. Treatment was given of 200 mgm. a day of α-tocopheral to these 29 patients. All of them had been on sulphone treatment for periods varying from 6 months to some years. Examinations of the hand conditions were made before and after α-tocopheral treatment, and it was found that 11 had experienced no change. One of these and the 18 other patients continued the treatment for another month, when it was found that in all of them there was more extension of the fingers and increase in the volume of the muscles, very marked in some of them. *Ernest Muir*

IRVINE, C. **The Running of a Small Leprosarium in Kenya.** *East African Med. J.* 1951, July, v. 28, No. 7, 280–82.

This article is described by its author as a "rigmarole, popular rather than scientific". However, it contains much practical wisdom and common sense. The author's method of administering sulphone treatment is primitive, but suited to simple people on the slopes under Mt. Kenya. "The patients . . . sit around in the shade of a big tree . . . and one after another is called, says how he is and how much sulphone he wants—' I'm feeling bad and I stopped two days ago ; put me up half a tablet '. Some are ambitious and need to be restrained. Others are the opposite and having had a reaction are disinclined to start again". After describing former difficulties he says "but with the advent of sulphones all that is changed, and it is the simplest thing to run a small hospital for leprosy patients now that they normally feel so well and in such good heart and so keen to treat themselves . . . The leprosy work could be run by an intelligent dresser almost entirely. An out-patient clinic of many hundreds could be carried on as an appendage to a general hospital." [DDS is apparently not the dangerous drug it was supposed to be, at least not in East Africa.]
Ernest Muir

DOULL, J. A. **The Control of Leprosy.** *Ann. New York Acad. Sci.* 1951, Mar. 30, v. 54, Art. 1, 134–41.

This is a clearly reasoned and expressed summary of our knowledge of the factors which govern the spread and control of leprosy at the present time. The frequence of sub-clinical infections is not known and we have no test similar to the tuberculin test in tuberculosis which might reveal it. Attention is directed to the complexity of the prevalence rate, which is actually "the incidence rate of the disease for a given unit of time multiplied by the duration of the disease expressed in the same unit . . . Because of the usual long duration and the usual low prevalence rate the actual attack rate of the disease must be very low". In a local community leprosy usually occurs in clusters of households. "We find that in households in which a lepromatous case occurs the attack rate is six times to eight times that observed in other households. Since the annual average incidence of any community is low, it follows that there are large sections in which the disease probably could not continue were it not for the spread of infection from the clusters or foci which have a higher transmission rate." A review of the salient points indicates that the "biological balance between man and the bacillus is at a critical level at present and that the scales may be tipped in favour of man by shortening the average period of patient-

infectiousness. Whether this can be accomplished with available drugs is a matter of conjecture. There is strong reason to believe that this might occur, provided that the campaign was directed accurately."

Some of the practical requirements for control by chemotherapy are listed : adequate staff and proper organization for the distribution of sulphones ; leprosy surveys to ascertain the prevalence of the disease ; if the objective is control of the disease, only open cases should be treated [nothing is said about cases likely to become infectious in a short time], districts and clusters of households with higher prevalence should be selected for first attention, and within these districts patients who are exposing young children to risk of infection ; a plan of control should envisage intensive efforts for many years.

This paper should be carefully studied in the original by all concerned with the control of leprosy. *Ernest Muir*

CARPENTER, C. M. **Murine Leprosy—its Usefulness as an Experimental Infection.** *Ann. New York Acad. Sci.* 1951, Mar. 30, v. 54, Art. 1, 101–6. [21 refs.]

The bacteriological, pathological and clinical resemblances and differences between the diseases caused by Stefansky's and Hansen's bacilli are discussed. With the development of new drugs for the treatment of leprosy a need arose for their rapid evaluation. By treating experimental rat leprosy, comparatively rapid screening of drugs can be carried out in 6 months to one year, where it would take 3 to 5 years in man. Carpenter and his colleagues [this *Bulletin*, 1950, v. 47, 52] found that promin and diasone failed to cure the disease in rats ; Promin suppressed the infection and increased the longevity of treated rats, whereas diasone did not significantly alter the course of the disease.

Another use of *Myco. leprae murium* is in testing for procedures for the isolation of this organism and *Myco. leprae.* *Ernest Muir*

HELMINTHIASIS

KOCH, W. & STEINITZ, H. **Multiple Helminthic Infections.** *J. Parasitology.* 1951, June, v. 37, No. 3, 307–8.

In a survey of a Jewish population, 147 Jews of Mediterranean or Asiatic extraction were found to have a higher rate of helminthic infection (56 per cent.) than 303 Jews of Eastern European extraction (25 per cent.). The rate among females (45 per cent.) was higher than that among males (35 per cent.). If the probabilities of infection by *Ascaris lumbricoides* and by *Trichuris trichiura* were independent, the number of people infected by both worms could be predicted by multiplying together the two individual infection rates. The predicted number of doubly infected people is 20, as compared with an observed value of 40. This result conflicts with those of other workers ; the authors are unable to suggest a reason for the discrepancy. *P. Armitage*

See also p. 1107, BONILLA NAAR & RODRÍGUEZ VELANDIA, Amibiasis autóctona en Bogotá (2,640 Mts.) e incidencia de parásitos intestinales en la clase media. (Parasitismo intestinal y eosinofilia.) [**Autochthonous Amoebiasis in Bogotá and Intestinal Parasitism in the Middle Classes. Eosinophilia and Intestinal Parasitism.**]

ERRATUM

In the abstract of the paper relating to schistosomiasis of the bladder in Somaliland, published in the May 1951 issue of this *Bulletin*, p. 477 and stated to be by EGIDIO, L., the author's name should read LIPPARONI, E.

In the same abstract the words in the first and second lines of the second paragraph " 40 cases of schistosomiasis of the bladder have been demonstrated in two years " should read " a monthly average of 40 cases of schistosomiasis of the bladder has been demonstrated in the last two and a half years ".

LAPIDUS, M. Uréthrite chronique et bilharziose vésicale. [**Chronic Urethritis and Vesical Schistosomiasis.**] *Maroc Méd.* 1951, Aug., v. 30, No. 315, 729.

An account of 2 cases.

GILGES, W. **Skin Polyp—only Symptom of Bilharzia Infection.** *East African Med. J.* 1951, July, v. 28, No. 7, 288.

An African girl of 8, seen in Fort Jameson, Northern Rhodesia, had a small polypoid growth on the pubic region just above the clitoris. The growth was 1 in.×½ in., stalked, soft and situated at the tip of a small depigmented area. Similar depigmented areas were seen on the abdominal and chest walls.

The polyp was removed and the wound healed by first intention. Histological examination proved the growth to be a skin polyp containing numerous schistosoma ova. Apart from the presence of hookworms in the stool, no abnormalities were found in stool or urine specimens and daily examinations for a week failed to reveal any schistosoma ova.

A leucocyte count a month later showed the presence of 11,500 leucocytes per cmm., of which 7 per cent. were eosinophiles. Temperature and pulse were normal while the patient was in hospital and no abnormalities were found on physical examination (but a cystoscope was not available).

H. J. O'D. Burke-Gaffney

BARRETO, Z. P. & ACCIOLY, J. Hematopenia esplénica esquistossomótica. (Contribuição ao estudo do hiperesplenismo.) [**Haematopenia of Splenic Origin in Schistosomiasis Mansoni.**] *Rev. Brasileira Med.* Rio de Janeiro. 1951, June, v. 8, No. 6, 395–8, 2 graphs. [22 refs.]

By the term haematopenia the authors would seem to imply leucopenia affecting all types of leucocytes and reduction of platelets, with little if any fall in erythrocytes. This condition may occur in infestation by *Schistosoma mansoni* associated with enlargement of the liver and spleen. The cause of the blood changes is thought to be the spleen by its action on the bone marrow, possibly hormonal. Two cases are detailed in which this condition was present ; one in a man of 36, the other in a woman of 20 years. In each splenectomy was performed and the response was rapid, a large advance towards restoration to normal being obvious within 2–3 days. The leucocytes rose from 3,250 per cmm. to a little over 14,000 in 3 days and were 10,150 a month later in the first patient ; from 3,600 to 8,100 in a month in the second patient. The platelets increased from 35,622 five weeks before the operation [no count was made nearer the time] to 101,216 on the third day after and 144,325 a month later in the first patient ; from 23,010 before operation to 252,462 a month later in the second.

H. Harold Scott

KOCH, K. R. & KUX, P. Die Verträglichkeit von Miracil D bei der Schistosomiasis mansoni in Brasilien. [**The Suitability of Miracil D in** *S. mansoni* **Infections in Brazil.**] *Ztschr. f. Tropenmed. u. Parasit.* Stuttgart. 1951, June, v. 3, No. 1, 94–100.

The investigations recorded in this paper were undertaken in Acesita in the State of Minas Gerais. The town has now 8,000 inhabitants, drawn from all parts of Brazil. In the neighbourhood there are many foci of schistosome infection, although *Australorbis* is absent. Those patients who were afflicted with *Schistosoma mansoni* must therefore have contracted the infection elsewhere, and the possibility of reinfection after apparent cure could be discounted. For treatment with Miracil D, patients were taken without selection and some had undergone previous treatment with antimony tartrate or other antimony preparations. It was found that Miracil D, in a daily dosage of 0·6 gm. or over, evokes in adult Brazilians with schistosomiasis toxic symptoms of such severity that the course of 5 days' treatment could not be completed.

The side-effects can be decreased by starting a course of 7–10 days' duration with one dragée (0·2 gm.) on the first day and by increasing by one dragée daily until the maximum daily dosage (about 5 dragées) is reached. This maximum is then maintained for some days and thereafter decreased by one dragée daily. The drug should be taken after meals.

Patients of 60 kgm. received a total up to 6 gm. of Miracil D ; those of 50 kgm. received 5 gm. and so on.

This scheme of varied daily dosage eliminates to a considerable degree the nervous symptoms. The gastrointestinal disturbances which are apt to supervene in the second half of the course are relieved by the intake of small amounts of food between meals.

The results have been followed up 3–6 months after cessation of treatment. Of 9 patients followed for 4 months, one remained positive, and of 7 after six months all were negative to stool examination for *S. mansoni* eggs. In many instances a follow-up was not possible. *P. Manson-Bahr*

DÍEZ MELCHOR, F. Esplenomegalia egiptiana. [**Egyptian Splenomegaly.**] *Med. Colonial.* Madrid. 1951, Sept. 1, v. 18, No. 3, 199–212. [54 refs.]

A general review.

KAZZAZ, D. S. & TABBARA, R. **The Effect of Repodral (Fuadin) on the Electrocardiogram of the Dog.** *Amer. J. Trop. Med.* 1951, July, v. 31, No. 4, 510–13, 2 figs.

" The intramuscular administration of Repodral in doses of 0·5 cc. per kilo (4·25 mgm. of antimony per kilo) over a two days period to normal dogs produced no significant changes in the electrocardiogram of these dogs taken under ether anesthesia."

HASKINS, W. T. **Determination of Sodium and Copper Pentachlorophenates in Dilute Aqueous Solutions.** *Pub. Health Rep.* Wash. 1951, Aug. 17, v. 66, No. 33, 1047–51.

" A rapid method of determining sodium and copper pentachlorophenates in concentrations of 1 to 100 ppm. in water is given. The method is intended for use in the field as an aid in investigating the molluscacidal action of these compounds in natural waters and involves the use of a minimum of equipment and reagents."

KOMIYA, Y. & ITO, J. **Contribution to the Morphology of** *Paragonimus westermanii*. *Japanese Med. J.* 1950, Oct., v. 3, No. 5, 309–14, 6 figs.

MORELL CUÉLLAR, L. & GONZÁLEZ CASTRO, J. Nuevo caso de distomatosis humana por *Fasciola hepática*. [**A New Case of Infection with** *F. hepatica*.] *Rev. Ibérica de Parasitología*. 1951, July, v. 11, No. 3, 271–81. [39 refs.]

The English summary appended to the paper is as follows :—

"It is described a new case of human Fasciolosis in Spain giving report of the new ones after 1946 in our country and abroad."

CHAVARRÍA C., M. *Fasciola hepática*, anomalías en el aparato reproductor femenino. [**Anomalies in the Female Reproductive Organs of** *Fasciola hepatica*.] *Rev. Med. Vet. y Parasit.* Caracas. 1950, Jan.–Dec., v. 9, Nos. 1/4, 69–83, 19 figs.

PAUTRIZEL, R., RIVASSEAU, J. & RIVASSEAU, D. Injection d'extrait de douve et éosinophilie sanguine chez le mouton douvé. [**Injection of Extract of a Liver Fluke and the Blood Eosinophilia following it in an Infected Sheep.**] *C. R. Soc. Biol.* 1951, May, v. 145, Nos. 9/10, 720–21.

The following is a translation of the authors' summary :—

The injection of extracts of *Fasciola hepatica* into sheep infected with this parasite provoked an intense fall in eosinophiles followed some hours later by a considerable increase in them. *H. J. O'D. Burke-Gaffney*

ALÉS REINLEIN, J. M., ARJONA TRIGUEROS, E. & OBRADOR ALCALDE, S. **The Study of Cerebrospinal Fluid in Diagnosis of Cysticercosis of the Central Nervous System.** *Bull. Inst. Med. Res.* Madrid. 1951, Apr.–June, v. 4, No. 2, 65–76. [26 refs.]

The authors state that cysticercosis and cysticercosis of the brain are commoner than is supposed, and that it is "extremely generalised" in North and South America and especially in Mexico and Chile. They quote literature in support of this statement. During the last two years 10 cases of cerebral cysticercosis in one of which the cysticercosis was generalized have been recorded at the Institute of Neurosurgery and the University Clinic at Madrid.

The authors discuss the changes in the cerebrospinal fluid in cerebral cysticercosis, quoting the relevant literature. The fluid looks normal or slightly opalescent. Its pressure is high. The discovery in it of cysticercal vesicles or membranes is rather exceptional. A pleocytosis of 5 to 500 cells per ml. [*sic.* presumably cmm.] may be present, the cells being mostly lymphocytes, large mononuclears, some polymorphonuclear neutrophiles and plasma cells. Eosinophiles are typical of the infection, eosinophilia varying between 0·5 to 72 per cent. In the 10 cases studied by the authors it varied between 2 and 42 per cent. It is not limited to cerebral cysticercosis and may be absent. Its presence led the authors to suspect cerebral cysticercosis and they consider that, when it is combined with a fall in the glucose level of the fluid, it is even more suggestive. Eosinophilia of the blood, on the other hand, is not frequent. Globulin and protein reactions and colloidal curves of the cerebrospinal fluid are not in the least specific of the disease. The Wassermann reaction is usually negative. The present authors never obtained a positive Wassermann, but others have found unstable positives. These may lead to a diagnosis of neurosyphilis.

Earlier work on the complement deviation reaction, and the varying opinions about its value for the diagnosis of cysticercosis, are discussed. The authors

used, for studies of their 10 cases of cerebral cysticercosis, an antigen made from pig cysticerci prepared by a modification of Lange's technique. The results were, they consider, excellent. They are given in a table, together with the results of their other observations on the 10 cases. In 7 of the 10 cases histological examination of surgically-excised tissue verified the existence of cysticerci ; operations were not done on the other three cases. One case showed generalized cysticercosis. Complement deviation was positive in all but 2 of the 10 cases, one of these 2 being negative ; the other could not be verified, but operation revealed the presence of cysticerci. The authors do not doubt the value of complement deviation, but they do not regard it as infallible. They quote literature showing that a negative reaction does not necessarily indicate the absence of cysticerci. One of their own cases always gave negative results, although different antigens were used, but operation revealed the presence of small numbers of cysticerci, as it did in the case whose complement deviation could not be verified. The results of the complement deviation reaction depend on the degree of infection and the location of cysticerci in the ventriculo-arachnoid ducts. The absence of changes in the cerebrospinal fluid may be due to the presence of cysticerci at distant sites, where they may be numerous, so that the lumbar, cisternal and ventricular fluids should all be examined if possible.

The authors' discussion of the cross reactions that may occur when antigens made from *Cysticercus cellulosae* and from hydatid cysts are used is, in places, somewhat obscure and must be read in the original, which is written in English. They conclude that antigen made from *Cysticercus cellulosae* is more selective and that, apart from clinical symptoms, an eosinophilia of the cerebrospinal fluid, especially if it is combined with a fall in the glucose level of this fluid, is suggestive of cerebral cysticercosis. The suspicion can be confirmed by complement deviation reactions. *G. Lapage*

HOEKENGA, M. T. **Treatment of *T. saginata* and *H. nana* Infestations with Atabrine.** *Amer. J. Trop. Med.* 1951, July, v. 31, No. 4, 420–22.

" 1. Atabrine hydrochloride, administered in a dose of 0·8 Gm. orally to 35 patients with *T. saginata*, caused the passage of a tapeworm in 32 instances. In 24 of the 35 (68·5 per cent) the scolex was found.

" 2. Atabrine hydrochloride administered in the same dose orally to 5 children with *H. nana* was not particularly effective, causing only temporary disappearance of ova from the stools.

" 3. There were no serious toxic reactions to the use of atabrine in the dosage described. In 8 cases (20 per cent) there was nausea and vomiting ; in 3 instances the vomiting was marked enough to cause failure of the treatment.

" 4. Atabrine is, therefore, a valuable teniafuge. It is easily administered, can be repeated more quickly than oleoresin of aspidium if it is necessary to do so, and it causes only transient toxic reactions."

HIRTE, W. Bandwurmkuren mit einem Zinnpräparat. [**The Cure of Tape Worm Infestation by a Preparation containing Tin.**] *Deut. med. Woch.* 1951, Aug. 31, v. 76, No. 35, 1083–5. [30 refs.]

A description of the treatments in common use for the eradication of tapeworms introduces the subject of tin as a therapeutic agent. Tin was used as a vermifuge in the days of Paracelsus and its successful use in Germany, England and Italy was later described by a number of writers. It fell into disrepute perhaps because the metal frequently contained arsenic and also

lead. The availability of pure tin again makes its use possible. Here described is a stable tablet preparation called Cestodin made by the Nematodin-Gesellschaft, Hamburg-Bahrenfeld, which contains metallic tin and also its oxide and chloride. It has no toxic or undesirable effect on patients and is very effective. In 58 cases, there were only 2 recurrences after 3 months. A tablet is given 3 times on the first and 6 times on the second day and then 1 tablet after each of the 3 main daily meals for 3 days. No purgatives are given but a dose of magnesium sulphate is given before, and one after, the treatment. There appear to be no contraindications to this therapy.

[The proportions of the constituents of the preparation are not stated.]

M. E. Delafield

IBOLEÓN HURTADO, E. Hidatidosis pulmonar en Granada. [**Pulmonary Hydatid Disease in Granada (Spain).**] *Rev. Ibérica de Parasitología.* 1951, July, v. 11, No. 3, 307–22, 7 figs. [32 refs.]

LARIZZA, Paolo & VENTURA, Sandro. L'Anemia da Anchilostoma. Studio patogenetico. [**Pathogenetic Study of Ankylostome Anaemia.**] Prefazione def Prof. Paolo INTROZZI. pp. ix+206, 30 figs. & 2 coloured pls. 1950. Pavia: Tipogr. del Libro. (Biblioteca "Haematologica" 10.) [22*s.* 6*d.*]

In this monograph, Larizza and Ventura report on their investigations in 112 cases of proved hookworm infection, which they have seen in the Medical and Pathological departments of the Pavia Medical School in the 10 years 1939–1948. The authors emphasize that, though legislation and other sanitary measures may have controlled the incidence of hookworm disease in workers employed at tunnelling, mining and brick-making, this helminthic disease is spreading in Italy among land workers, for whom it is more difficult to enforce the necessary preventive standards of sanitation and public health control.

After giving a few historical notes, the authors discuss the several different theories which have been elaborated to explain the aetiology of hookworm anaemia. The supporters of the "mechanical" theory first believed that hookworm fed on the blood of its host, and that anaemia in the latter was directly due to such blood loss. When it was shown that the main diet of the hookworm was the intestinal mucus and not the blood of the host, it was believed that the loss of blood responsible for the anaemia was due to the wound left on the intestinal wall by the hookworm and also to anti-coagulants produced by this parasite. The "toxic" or "toxic haemolytic" theory is then discussed and discarded by the authors who are in favour, with CRUZ, of the "deficiency" theory, by which depletion and insufficient replacement of iron are taken to account for hookworm anaemia—a "long lasting nutritional deficiency" being the main factor, with "the larger elimination of iron through the haemorrhages and the exaggerated mucus secretion of the intestine" as a secondary factor in the aetiology. It is stressed that diminished absorption of iron by the bowel is not strictly an aetiological factor in hookworm anaemia.

Of the 112 cases of hookworm infection which are dealt with, 84 (75 per cent.) showed blood changes, and a detailed account is given of the results of exhaustively investigating many specimens of blood and bone marrow in each case, with graphs and tables of findings; two coloured plates show the microscopic picture of blood and bone marrow before and after iron therapy.

A whole chapter (30 pages) deals with the metabolic exchange of iron in hookworm disease and there are diagrammatic representations of the whole complicated process in which loss of iron accompanies the loss of blood in the

host, calls for replacement and consequent rise in production of red blood corpuscles which in turn leads to over-consumption of iron ; the increased activity of the gastro-intestinal mucosa modifies the digestion of the organic iron in the food.

In another chapter the metabolism of the ferro-proteins through the tissue cells is dealt with.

The metabolism of riboflavin and lactoflavin has a chapter to itself, and so have the metabolism of copper and of the lipids.

Anaemia does not occur in every human case of hookworm infestation ; it supervenes when the reserves of iron in the body have been depleted beyond such critical low limit of the "stock pile" that this can no longer be made up by an adequate absorption of more iron from the food. If this iron deficiency becomes established, disinfestation alone may fail to lead to recovery from the anaemia ; on the other hand, the anaemia may be cured by appropriate means which do not necessarily include complete riddance of the parasite.

This monograph is a specialized work which should prove useful in the study of hookworm anaemia as it occurs in other parts of the world.

J. Cauchi

CARVALHO, A. de A., BRAGA, N. de P., STRAUSS, Annelise, ARMINANTE, Hedda, ALTENHEIN, Denise, ROCHA, H. S. & VIEIRA, P. B. Anemia ancilostomótica na criança. [**Hookworm Anaemia in Children.**] *Arquivos de Clínica.* Rio de Janeiro. 1951, Mar., v. 12, No. 3, 216–24, 5 figs.

It is the belief of many that the anaemia associated with ankylostomiasis is due to iron deficiency resulting from intestinal haemorrhage caused by the parasites, and alimentary deficiency which reduces the amount of iron which should be present in the food. This may be so in the adult patient but it does not altogether hold good in children. The authors quote cases to show that careful dieting and administration of iron and blood transfusion, though bringing about a certain degree of improvement, will not cure so long as the worms remain. For example, a boy of 5 years of age, after 4 doses of tetrachlorethylene, was still passing 42,000 ova of hookworms per gramme of faeces ; his red cells were down to 500,000 per cmm., Hb. 1·5 gm. per cent. He was given Ferri et ammon. cit. and iron sulphate and had 21 transfusions, totalling 3,600 ml. After 6 months the improvement was only partial, red corpuscles 3,000,000, Hb. 8·7 per cent. (at 2 months these figures had been 2,100,000 and 6·8) and he was still excreting 26,000 ova per gm. of faeces. In 10 months, having had 14 doses of the anthelmintic, his blood showed 4 million red cells per cmm. and Hb. 11·9 gm. per cent., and there were no ova nor sign of blood in the stools. In other words, it was not until the worms had been got rid of that the anaemia cleared up. [It is understandable that haemorrhage would continue so long as worms are present. The authors do not discuss the possible toxic element which might play a part in causing the anaemia and which would cease to act when the worms were eliminated.] *H. Harold Scott*

GONZÁLEZ CASTRO, J. Hallazgo de *Metastrongylus apri* (Gmelin 1790) Vostokov 1905, en los pulmones de un niño. [*M. apri* **in the Lungs of a Child.**] *Rev. Ibérica de Parasitología.* 1951, July, v. 11, No. 3, 265–70, 5 figs. on 1 pl.

The English summary appended to the paper is as follows :—

"It is recorded a very first case in Spain of likely pulmonary parasitism in a child by *Metastrongylus apri* (Gmelin, 1790)."

MÜLLER, Gertrud. Zur Frage der Widerstandsfähigkeit von Askarideneiern gegen Druck. [**The Resistance of *Ascaris* Eggs to Pressure.**] *Gesundheits Ingenieur.* 1951, Aug., v. 72, No. 16, 281–2.

It has been suggested that exposure to pressure followed by its sudden release kills *Ascaris* eggs. This paper deals with experiments undertaken to test this view, for if it were found to be true, it might obviate the necessity for some purification of sewage containing *Ascaris* eggs prior to its disposal on land intended for growing crops for human beings.

Ascaris eggs in sewage were counted by microscopical enumeration of eggs filtered off by a membrane filter. Sewage was again examined after having been submitted to pressures of up to 4·5 additional atmospheres in the pumps used for distributing it on to land.

No effect as regards appearance and viability of the eggs could be demonstrated as the result of this pressure. *M. E. Delafield*

PAGÈS, R. & GAUD, J. A propos de la filariose au Maroc. Un cas de parasitisme humain par gongylonème. [**Infection by *Gongylonema* in Morocco.**] *Maroc. Méd.* 1951, June, v. 30, No. 313, 584–5. [16 refs.]

[See this *Bulletin*, 1951, v. 48, 667.]

CHABAUD, A. G. & LANZ, P. Pseudo-parasitisme de l'homme par *Agamomermis* sp. [**Pseudo-parasitism of Man by *Agamomermis* sp.**] *Ann. Parasit. Humaine et Comparée.* 1951, v. 26, No. 4, 376–8, 1 fig.

The authors describe a nematode removed by a village chief of the Kwango district of the Province of Léopoldville, Belgian Congo, from the urethra of his daughter, aged 10. The child had had, during the night, violent hypogastric pain and the next morning the worm had appeared at the meatal orifice. When she was seen three days later, the child still had a very few red blood corpuscles in the urine and an eosinophilia of 5 per cent. There is no schistosomiasis in the district. The nematode, which was badly preserved, was classified by the authors as a small specimen of *Agamomermis restiformis.* This is a genus of the Mermithidae, the adult phases of which are non-parasitic in the soil, the larval phases being parasitic in the body cavities of insects. The authors refer to the 5 other cases of infection with Mermithids known to them. Two of these were accidental infections of the digestive tract and three were cases in which the nematodes were found in the urethra, the case here recorded being a fourth in this latter category. The authors agree with the view that Mermithids found in the urethra have always been introduced voluntarily by the patient. *G. Lapage*

BASNUEVO, J. G., COWLEY CHÁVEZ, O., SOTOLONGO, F., BLANCO RABASSA, E. & ACHKAR, R. Tratamiento de la tricocefaliasis por los enemas de Hexilresorcinol llevados hasta el ciego. [**Trichuriasis treated with Enemata of Hexylresorcinol reaching to the Caecum.**] *Rev. Kuba Med. Trop. y Parasit.* 1951, Mar.–Apr., v. 7, Nos. 3/4, 48–50, 1 fig. [11 refs.]

The authors' mode of procedure is as follows : the day before treatment with hexylresorcinol is begun an evacuant enema is given at 9 p.m. and another at 6 a.m. the next morning. At 9 a.m. an emulsion of barium sulphate, 10 per cent., is prepared, and hexylresorcinol added to make 1 in 300. This is introduced into the rectum by a No. 22 rubber catheter passing some 15 cm. into the rectum. The patient then turns to the right to facilitate the enema reaching the caecum and then to the prone position and the filling is confirmed by the

fluoroscope. The quantity of the preparation needed to reach and fill the caecum is thus determined. Subsequent enemata consist of hexylresorcinol 1 in 300 in 5 per cent. gum acacia at 37°C., given every two or three days and retained for 10–30 minutes. The average amount is 15 cc. per lb. body weight ; so for a child of 40 lb. the amount would be 600 cc. Usually three to five enemata are given altogether.

If ova are still seen in the faeces a week after the course is finished, the drug is given by duodenal sound ; to adults 1·5 gm. hexylresorcinol in 300 cc. of 2 per cent. gum acacia emulsion ; to children 0·15 gm. in 30 cc. of the acacia for each year of age. From the age of 10 years the adult dose is given. The patient should have no food the day of the operation and only fluid—milk, soup, fruit juices—the day before. No purgative is needed, either before or after.

In some cases a mixture of hexylresorcinol and tetrachlorethylene is given by intubation, 1 gm. of the former, 4 cc. of the latter and 25 cc. of peanut oil (*aceite de mani*) suspended in 2 per cent. acacia. The dose is 30 cc. for adults, 3 cc. for each year of a child's age, and after 10 years the adult dose.

If the case still proves refractory, the authors prefer to have recourse to pentavalent arsenicals [see this *Bulletin*, 1949, v. 46, 388].

H. Harold Scott

DEFICIENCY DISEASES

LENELLE, J. & PARENT, M. Contribution à l'étude de l'état de nutrition du noir au Katanga. [**Study of the Nutritional State of Africans at Katanga.**] *Ann. Soc. Belge de Méd. Trop.* 1951, Apr. 30, v. 31, No. 2, 251–87, 10 graphs.

An account is given of the nutritional state of the people in a part of the Belgian Congo during the years 1948 and 1949. It includes an analysis of causes of death, records of heights and weights of schoolchildren, the incidence of signs of vitamin deficiencies and haematological and parasitological surveys. Widespread evidence of malnutrition was provided by the frequency of vitamin deficiencies, and in two groups of children only 6 per cent. were without clinical signs of malnutrition.

R. Passmore

LAL, S. B. **Dietary Habits of Depressed Class (Chairo and Dusadh) in Palamau.** *Indian J. Med. Res.* 1950, Oct., v. 38, No. 4, 369–75.

Twenty families in each class were surveyed over a period of ten days in September, 1947. The economic level of these Bihari peasants was very low and consequently their diet was poor. Thus, average daily consumption per head was only 1,760 and 2,140 calories. Milk and fresh vegetables were taken in very small amounts.

R. Passmore

LUYKEN, R. **Problems of Nutrition in Java and the Surrounding Islands, 1946–1950.** *Documenta Neerlandica et Indonesica de Morbis Tropicis.* Amsterdam. 1951, June, v. 3, No. 2, 126–46. [26 refs.]

After the defeat of the Japanese the Netherlands Red Cross sent a " Feeding Team " to Java, and the admirable report of the team has already been reviewed [this *Bulletin*, 1950, v. 47, 511]. In June, 1946, the greater part of the team returned to Holland and the remainder joined the Institute of Nutritional Research, Djakarta, which had resumed its activities. The present paper is a summary of the work of the Institute during the subsequent three years.

Owing to the chaotic condition in the surrounding countries and islands, survey work was greatly hampered. "Light symptoms" of deficiency disease were common. The deficiencies were the same as before the war, especially of protein and vitamin A. The chaotic conditions during and after the war added to these a deficiency of calories. A great increase in the incidence of tropical diseases contributed to the unfavourable state of nutrition. Diets in hospitals, institutes and messes often left much to be desired. Children were often stunted in growth.

In the laboratory some routine analyses of food were carried out. In a preliminary experiment it was shown that a diet containing Javanese maize retarded the growth of white rats, but that this could be remedied by nicotinic acid or tryptophane or both. *R. Passmore*

Erratum

In the abstract of the paper relating to the diet of the Somali in relation to health, published in the June 1951 issue of this *Bulletin*, p. 578, and stated to be by Egidio, L., the author's name should read Lipparoni, E.

Schlesinger, P. & Benchimol, A. B. Coração beribérico simulando cardiopatia arteriosclerotica. [**Cardiac Beriberi suggesting Arteriosclerotic Heart Disease.**] *Arquivos de Clinica.* Rio de Janeiro. 1951, Jan., v. 12, No. 1, 1–11, 5 figs. [10 refs.]

The English summary appended to the paper is as follows :—

"A report is presented of two fatal cases of cardiac beri-beri, in which the clinical diagnosis was considered improbable due to a set of circumstances suggesting arteriosclerotic heart disease. The first patient, although a young adult, showed left bundle branch block in the electrocardiogram, whereas the second, a 52-year-old male, complained of paroxysmal dyspnea as the initial symptom with electrocardiographic abnormalities indicating left ventricular hypertrophy and myocardial damage. However, a history of chronic alcoholism, the presence of polyneuritis and especially the post-mortem examination, ruled out the diagnosis of coronary sclerosis. The cardiac changes revealed at autopsy were of the type which has been frequently described in beri-beri heart disease, namely interstitial edema and degeneration of the myocardial fibres. In the absence of any other etiologic factor these changes were sufficient to establish that vitamin deficiency was the evident cause of heart disease in these patients. It seems therefore, that these cases illustrate the possibility of cardiac beri-beri leading to advanced degrees of myocardial fibrosis which determine electrocardiographic changes indistinguishable from those observed in coronary sclerosis and other types of degenerative myocardial disease."

Winckel, W. E. F. **Changes of the Myenteric Plexus in Pellagra.** *Documenta Neerlandica et Indonesica de Morbis Tropicis.* Amsterdam. 1951, June, v. 3, No. 2, 100–104, 3 figs. [12 refs.]

An account is given of the post-mortem examination of a prisoner of war who died of pellagra in Siam. Most of the findings were already well known, but the degenerative changes in Auerbach's plexus, both in the jejunum and ileum, are not generally described.

"The more or less oval nucleus of the ganglion cells was generally localised near the wall and its chromatin pattern was either too distinct or there was hyperchromatosis of the nuclear wall. Ganglion cells with pyknotic nucleus and even dark-coloured protoplasm were surrounded by a too large number of

satellite cells with rather large oval nuclei. Polynuclear giant cells had often developed from these satellite cells ; they lay against the cellular body of the ganglion cell, often ' denting ' the protoplasm." *R. Passmore*

JELLIFFE, D. B. **Clinical Rickets in Ibadan, Nigeria.** *Trans. Roy. Soc. Trop. Med. & Hyg.* 1951, Aug., v. 45, No. 1, 119–24, 4 figs. on pl. [14 refs.]

Rickets is generally considered to be a rare disease in the tropics. In recent years reports by some observers tend to show that this view is probably incorrect, MANSON-BAHR [this *Bulletin*, 1946, v. 43, 607] and ROGERS and MEGAW [*ibid.*, 1942, v. 39, 282] being of this opinion. Little appears to have been written on the subject of vitamin D deficiency in Africa, and the author studied a series of cases in Ibadan, Nigeria.

Diagnosis was made on clinical grounds only, radiological examination and estimation of blood phosphatase not being performed. The five clinical signs especially looked for were : (1) closure of the anterior fontanelle, (2) shape of the head, (3) craniotabes, (4) enlarged epiphyses, (5) enlarged costo-chondral junctions. In addition such other evidence as marked delay in walking, bow-legs, etc., was noted.

Ibadan is a large insanitary city of about half a million inhabitants, and the majority of the inhabitants are Yoruba. Two hundred children between the ages of 3 months and 3 years, mostly of poor parents, were observed in the out-patient department between January and April, 1950. Of 50 children, aged about 2 years, the anterior fontanelle was found to be widely open in all, while in 35, aged about 3 years, it was open in 14 per cent. In three children, not included in this series, the anterior fontanelle was definitely open at 3½ years of age, and in one case closure was not complete by the 4th year.

A square, enlarged, box-like skull (*caput quadratum*) was found in 20 per cent. of cases. Bossing was present in 17 per cent. of all cases. Two other causes of bossing of the skull had to be excluded, *viz.*, calcifying bilateral cephalhaematoma, and marked sickle cell anaemia. Craniotabes was found in 5 per cent. of children between the ages of 3 months and 1 year, and enlargement of the costo-chondral junctions in only 5 per cent. of all cases.

It is remarked that the clinical picture of rickets may vary in different parts of the world.

Eight instances of delayed walking, one of a child " going off his feet ", and one of spasmophilia were seen. Most of the group had a varying degree of respiratory tract infection, varying from a mild bronchitis to well marked broncho-pneumonia. Response to treatment was definite in a number of cases, but out-patient attendances tended to be irregular. Possible aetiological factors were :—

(*a*) Deficient maternal diet. The food of the poorer Yoruba woman rarely contains any animal protein and is almost certainly deficient in vitamin D. The effect of ultra-violet irradiation is probably largely counterbalanced by the fact that the town women tend to be much more heavily dressed than those living in the country.

(*b*) Prematurity (low birth weight). WALKER (1950, personal communication) found that in Ibadan the average birth weight in 704 full term infants was 6 lb. 5 oz., and this may possibly further increase the inadequate intra-uterine storage of vitamin D.

(*c*) Insufficient exposure to ultra-violet light. Children are usually over-clothed and spend the greater part of their early life strapped on the mother's back with a wide length of cloth.

(*d*) Dietary deficiency in infancy. It has been pointed out that too prolonged breast-feeding predisposes to rickets, and this may apply in Ibadan,

where the poorer children are breast-fed up to 18 months and, in extreme cases, up to 3 years of age. When weaned they are given a diet containing almost entirely carbohydrates, namely yams, cassava, maize. The last has a high content of phytic acid, which interferes with the absorption of calcium from a diet already deficient.

Cases of rickets seen in Ibadan are almost always mild and self-limiting, presumably because older children have an increasing amount of solar irradiation as they spend less time on their mothers' backs and crawl about with comparatively few clothes on. Except for an increased liability to respiratory infections little actual ill-health is caused in most cases, but occasional permanent structural deformities may result. Marked rachitic pelvis is rare. The author remarks that as a vitamin deficiency disease rickets has nothing like the immense importance of the various syndromes caused by lack of the vitamin B complex or of animal protein. This type of case could be easily prevented if children were weaned at an earlier age and put on a balanced diet, but for economic reasons this is at present impossible. Shark liver oil may become available in the near future from the shark fisheries which it is hoped to establish on the West African Coast. *C. F. Shelton*

CLARK, M. **Kwashiorkor.** *East African Med. J.* 1951, June, v. 28, No. 6, 229–36, 4 figs. on 2 pls.

This paper is a straightforward clinical description, based on observations of 310 cases at Fort Hall Hospital. It is recommended reading for anyone either going out to Africa for the first time or contemplating writing a textbook of general medicine or paediatrics. *R. Passmore*

VELIATH, G. D. & SRINIVASACHAR, S. **Cirrhosis of the Liver in Madras Province, South India.** *J. Indian Med. Ass.* 1951, May, v. 20, No. 8, 271–4, 5 figs. on pl. [16 refs.]

The clinical histories and post-mortem reports of 121 persons dying of portal cirrhosis in the three main hospitals in Madras Province are reviewed. Males outnumbered females by nearly 5 to 1 and the cases were evenly distributed from the second to the sixth decade of life. Just under half showed a histological pattern of fatty infiltration with varying degrees of fibrosis of the liver. On analogy with animal experiments these are classified as " deficiency cirrhosis ".

It is pointed out that nutritional defects form the most important aetiological factor and that in these cases the disease is not only preventable, but amenable to treatment in the early stages.

The remaining cases are classified as " post-necrotic cirrhosis ". The factors responsible are not known, but certainly are not malaria, amoebic or bacillary dysentery, but 20 per cent. gave a clinical history of a previous attack of jaundice. *R. Passmore*

BERGOUNIOU, J. L. Aperçu sur les cirrhoses nutritionnelles et la diététique à leur appliquer. [**Nutritional Cirrhosis and Diet Appropriate in its Treatment.**] *Bull. Méd. de l'Afrique Occidentale Française.* 1951, v. 8, No. 1, 31–6.

This paper is a short review of recent experimental and clinical work on hepatic cirrhosis due to nutritional factors.

The conclusion is that fatty degeneration is the essential lesion and is associated with a lack of lipotrope factors in the diet. The condition is aggravated

by an excess of carbohydrate or fat. It is logical therefore in treatment to give a diet rich in first-class proteins with limitation of salt to control oedema.

C. C. Chesterman

HAEMATOLOGY

BIGGS, Rosemary & ALLINGTON, M. J. **The Sampling Error in Haemoglobin Determination.** *J. Clin. Path.* 1951, May, v. 4, No. 2, 211–17, 2 figs.

The method of haemoglobin estimation that gives the most reliable results is that in which oxyhaemoglobin is estimated in the M.R.C. neutral-grey wedge photometer. The average standard deviation with this method is 1·6 per cent. of haemoglobin ; thus by this method differences in haemoglobin level of 5 per cent. would be considered significant. In practice this degree of precision is not reached because of the added errors of random sampling.

The first experiment was made in four blood samples, normal and anaemic ; 50 readings were made on each by five observers using different pipettes. The standard deviations varied from 1·28 to 2·04 and the average of the whole series was 1·59 per cent. ; the error was independent of the level of haemoglobin.

In the second experiment, 20 capillary blood samples were taken in rapid succession from a healthy person and were examined by two observers : the average standard deviation was now 4·5 per cent.

In the third experiment, the variations of haemoglobin readings on capillary blood samples taken on different days were estimated. For this 20 healthy subjects and 20 patients were examined, and from each, five samples were taken at the same time on six or seven successive days. Allowance was made for the trends that were apparent in the patients. Haematocrit values were also taken and were shown to run parallel to the haemoglobin readings from day to day. The extent of the observed variation was wide, ranging from standard deviations of 1·65 to 6·96 per cent. A table was calculated which shows the range of variation at different haemoglobin levels that may be expected for different counts in the same individual. In this table there is a column showing the differences that may be considered significant ; from 120 per cent. to 40 per cent. of haemoglobin, this figure varies from 10 to 13 per cent. and at the 30 per cent. and 20 per cent. levels it is 8 per cent. and 6 per cent., respectively.

The fourth experiment was designed to test the variability of venous and capillary samples from day to day ; samples were taken from five normal subjects on ten consecutive days. Random variations and day-to-day variations were calculated separately ; the latter showed a coefficient of variations of 4·40 per cent. for capillary blood, 4·62 per cent. for venous, 4·30 per cent. for the haematocrit, and only 1·91 per cent. for the M.C.H.C. It is recommended that the M.C.H.C. should always be calculated from readings obtained on a single sample of blood.

L. E. Napier

PERUTZ, M. F., LIQUORI, A. M. & EIRICH, F. **X-Ray and Solubility Studies of the Haemoglobin of Sickle-Cell Anaemia Patients.** *Nature.* 1951, June 9, 929–31, 3 figs.

See also p. 1136, CARVALHO, BRAGA, STRAUSS, ARMINANTE, ALTENHEIN, ROCHA & VIEIRA, Anemia ancilostomótica na criança. [**Hookworm Anaemia in Children.**]

See also p. 1135, LARIZZA & VENTURA, L'Anemia da Anchilostoma. Studio patogenetico. [**Pathogenetic Study of Ankylostome Anaemia.**]

See also p. 1131, BARRETO & ACCIOLY, Hematopenia esplénica esquistossomótica. (Contribuição ao estudo do hiperesplenismo.) [**Haematopenia of Splenic Origin in Schistosomiasis Mansoni.**]

DE CAMARGO, T. A Hematologia no interior do Brasil. Conceitos sôbre o diagnóstico diferencial de alguns tipos de esplenomegalias crônicas à luz do exame hematológico. [**Observations on the Differentiation of Various Types of Chronic Splenomegaly on the Basis of Haematological Examination, in Brazil.**] *Anais Paulist. Med. e Cirurg.* 1951, June, v. 61, No. 6, 379–403. [44 refs.] English summary.

VENOMS AND ANTIVENENES

FLOCH, H., BARRAT, R. & ABONNENC, E. L'envenimation par piqûre de scorpions en Guyane française. [**Scorpion Sting in French Guiana.**] *Institut Pasteur de la Guyane et du Territoire de l'Inini. Publication No. 219.* 1950, Nov., 4 pp.

Scorpion stinging is rare in French Guiana, probably because the population is sparse, 28,546 in the 86,000 sq. kilometres, or one to every 3 kilometres.

Three cases are recorded here. The symptoms vary considerably, but the commonest are hypersensitivity, vomiting, rapid breathing, salivation, profuse sweating, local pain, followed by cyanosis and muscular twitching. Other symptoms peculiar to these three patients were as follows :—

1. A man of 38 years, after bathing, sat on a "black scorpion" which naturally reciprocated. A quarter of an hour later he complained of numbness of the legs, followed by a general malaise, "electrical shocks" in the limbs and contraction of the jaw muscles ; later convulsions, syncope and semi-coma. He was bled, given an injection of physiological saline [quantity not mentioned] and of snake antivenene and camphorated oil. Improvement was rapid but he continued to suffer from vertigo and was not really well until 5 days later.

2. A sergeant in the nursing service, stung on the right little finger. He encouraged local bleeding and applied a tourniquet. An hour later, as the pain was only slight, he released the tourniquet and in a quarter of an hour his limbs felt numb and there was intolerable tingling and disturbance of vision. He was given an injection of anti-*Bothrops* and anti-*Crotalus* serum, without benefit, and in 7 hours another injection of antivenene was administered. He became intensely dyspnoeic with troublesome hiccough. Another 10 cc. of antivenene was given intravenously with camphorated oil. For some hours he felt very weak and vomited ; the disturbance of vision persisted until the following day, after which convalescence was rapid.

3. A man of 34 years, stung on the thigh ; he kept at work for half an hour, when, unable to walk he was taken to hospital by motor and on arrival was semi-comatose. He was cold, distressed and dyspnoeic, with frequent myoclonic movements and hiccough with profuse salivation. He, too, was given antivenene (not specified), physiological saline, 500 cc., camphorated oil and strychnine. His condition remained serious during that day but was better next day and he left hospital 4 days later.

The offending scorpion probably in each case and identified certainly in one was *Tityus cambridgei*, a large brownish-black scorpion, measuring 84 mm. in the case of the male, 72 mm. the female. It is common in Brazil, Dutch Guiana

and Panama. Other species met with in French Guiana are *Broteas gervaisii*, *B. granulatus*, *Brotochactas delicatus*, *Tityus stigmurus* and *Isometrus maculatus*.

The authors note the difficulty of obtaining specific antivenene in French Guiana and the value of subcutaneous injections of saline. [On this last, see this *Bulletin*, 1936, v. 33, 400 ; 1937, v. 34, 721.] *H. Harold Scott*

DERMATOLOGY AND FUNGUS DISEASES

LACAZ, C. da S. Lesões pulmonares na blastomicose sul-americana. Inquérito preliminar realizado com a paracoccidioidina. [**Pulmonary Lesions in South American Blastomycosis. Preliminary Investigation carried out with Paracoccidioidin.**] *Hospital*. Rio de Janeiro. 1951, Mar., v. 39, No. 3, 405–22, 5 figs. [26 refs.]

Pulmonary lesions were formerly believed to be relatively uncommon in South American blastomycosis (*Paracoccidioides brasiliensis*) but recent observations based chiefly on radiography of the chest and on careful necropsies have not supported this view. The present author, in a study of 35 cases of the disease, found pulmonary lesions present in 20 (57 per cent.).

Radiologically, the pulmonary lesions may be classified as (*a*) Nodular, including miliary, pseudo-tumoral and intermediate forms and forms with dissemination, (*b*) a fibrotic type with proliferating peri- or para-nodular fibrosis or with pleural thickening and (*c*) forms of cavitation.

Infection contracted by inhalation of spores of the fungus may give rise to a local lesion with inflammation and infiltration of the bronchioles and eventual development of a disease marked by cough, sometimes with blood-stained sputum, fever and emaciation which may simulate pulmonary tuberculosis. The infection may spread (*a*) directly on the mucous surface to the contiguous alveoli, or into the peribronchial tissues which become infiltrated and thickened and show interstitial changes, sometimes being the site of small abscesses, (*b*) by the blood stream, the fungus reaching the alveolar septa in which a granulomatous infiltration develops and extends to form nodules, (*c*) by the lymphatics, (*d*) by both the blood stream and the lymphatics, (*e*) through the pleural sac, and the rare case in which the lung tissue was infected by the penetration of a diseased fragment of a fractured rib.

The paracoccidioidin skin sensitivity test was performed with antigenic material prepared according to the method used by Smith for the preparation of coccidioidin. On 10 healthy people with no history of blastomycosis, the test gave entirely negative results, of 18 people with South American blastomycosis 10 reacted, of 18 people with dermal leishmaniasis 3 reacted but these 3 were also tuberculin-positive. Three out of 10 people with chromoblastomycosis also reacted and they were tuberculin-positive. In a group of 330 hospital patients suffering from various diseases, 26 (7·8 per cent.) reacted to the paracoccidioidin test and 25 of these were also tuberculin-positive. None of the 26 showed any radiological evidence of pulmonary disease. The interpretation of these results presents a difficult problem ; the specific quality of the antigen has not yet been proved and the possibility of cross reactions cannot be overlooked. Regression of the pulmonary lesions of Lutz's disease (South American blastomycosis) is assumed to occur and in such cases a specific allergic sensitivity might persist.

The author proposes to carry out another series of tests with a more dilute antigen capable of giving more specific results. [There seems no reason, from the evidence set out, to suspect that the unaccountable reactions are associated with sensitivity to histoplasmin.] *J. T. Duncan*

MISCELLANEOUS DISEASES

CARLE, R. Das Ökologische Mosaik der Infektketten bei einigen Seuchen in Südrussland (Tularämie, Pest, Malaria, Pappatacifieber). [**The Ecological Complex which determines the Chain of Infection in Tularaemia, Plague, Malaria and Sandfly Fever in South Russia.**] *Ztschr. f. Tropenmed. u. Parasit.* Stuttgart. 1951, Apr., v. 2, No. 4, 558–602, 16 figs. [Numerous refs.]

This paper is reviewed in *Bulletin of Hygiene*, 1951, v. 26, 1085.

FAUST, E. C. **Problems of Tropical Medicine in the United States.** Reprinted from *Thirty-Third Annual Meeting Medical Section, American Life Convention, Chicago, Ill., June 23, 1944.* 96–119, 9 figs. [20 refs.]

This is a war-time paper dealing with the history of a number of infectious diseases in the U.S.A., and with the possibility of introduction of disease by returning troops. Though the topicality is lost, some of the interest of the historical review remains, particularly that of the distribution of malaria, which is said to have been introduced from Europe. Its spread and peaks of incidence are described and illustrated by maps, one of which reconstructs the probable distribution of 1850 to 1860. The other diseases of which brief accounts are given include yellow fever, dengue, epidemic, murine and tick typhus, plague, cholera, the dysenteries, hookworm disease, filariasis, and leprosy. *G. Macdonald*

MONTEL, L. R. Un cas de *Larva migrans* provenant du Brésil. [**A Case of Larva Migrans Derived from Brazil.**] *Bull. Soc. Path. Exot.* 1951, v. 44, Nos. 7/8, 436–7, 1 fig.

PROTOZOOLOGY : GENERAL

HART, E. W., PAULLEY, J. W., RIVERS, J. S. & WESTLAKE, E. K. **Toxoplasmosis.** *Arch. Middlesex Hosp.* 1951, Jan., v. 1, No. 1, 26–50, 7 figs. (1 coloured on pl.) [41 refs.]

This is a comprehensive account of toxoplasmosis ; the morphology of the parasite, epidemiology, pathology, clinical picture, cardinal signs, and the clinical, radiological and serological diagnoses, and the treatment, are discussed successively. The paper gives one of the best accounts of the disease that has appeared in Britain and it is fully annotated ; there are 40 references to the literature.

There are six case reports included, two of which have been reported by SUTTON [this *Bulletin*, 1951, v. 48, 1047]. Of the remaining 4, one was a young man of 18 years who was admitted after losing consciousness. He gave an earlier history of convulsions, clumsiness of left hand since infancy, visual defects and frontal headaches. He showed flakes of calcification scattered through the right parietal area and two large calcifications deep in area of the basal ganglia, choroido-retinitis, and a positive complement-fixation test.

Another was a female of 23 years, sister of another patient with toxoplasmosis, with a history of Jacksonian epilepsy. She showed scattered cerebral calcifications but a negative complement-fixation test, and was probably a " burnt-out " case.

The fifth case was a child of 2½ years with microphthalmos and acute choroiditis, intracranial calcifications and positive serum test ; the mother and brother, both well, also had positive serum tests.

The sixth case (the only doubtful case of the series) was a child of 10 with defective vision and a squint : he had old choroiditis, small calcifications in the frontal and parietal lobes, but negative serological tests : the mother's serum showed a positive complement-fixation test in low titre. This was possibly a " burnt-out " congenital case.

Toxoplasma is a protozoon 4 to 7 μ by 2 to 4 μ, an obligate intracellular parasite living in fixed tissue cell, with a low degree of viability. It is found in many animal species. Its mode of transmission to man is unknown, but among other means droplet infection has been suggested. It produces proliferative granulomata occasionally with necrosis in many organs and tissues. The parasite spreads by the blood stream ; it invades the blood-vessel walls and extends from here into the surrounding tissue.

It has an affinity for nerve tissues where, apparently because antibodies are unable to reach it in sufficient strength, it persists when the infection is regressing in the other tissues. The nervous tissues of the young are more susceptible than those of adults.

Clinically it may be congenital or acquired. In the former case it may present as hydrocephalus *in utero*, as acute necrotizing encephalomyelitis of the newborn, or in a subacute or chronic form with the classical tetrad, convulsions, choroido-retinitis, cerebral calcifications and hydrocephalus.

The acquired infection may present as encephalitis in childhood, as acute exanthematic disease with atypical pneumonia or as an atypical encephalitis in adults, or it may be a latent sub-clinical infection.

Toxoplasma may rarely be identified during acute infection in the blood, marrow, cerebrospinal fluid, sputum or by biopsy. At autopsy it is found in many tissues. It may be grown on suitable medium or it may be identified by animal inoculation in mice, guineapigs or rabbits.

The serological tests include the neutralizing antibody test, the complement-fixation test, the complement-modifying test and the intradermal test.

L. E. Napier

RIEGER, H. Toxoplasmose. [**Toxoplasmosis.**] *Wien klin. Woch.* 1951, Sept. 7, v. 63, Nos. 35/36, 680–83. [42 refs.]

General review and discussion.

HOLDEN, W. S. & WHITEHEAD, A. S. **The Radiological Appearances in Congenital Toxoplasmosis.** *Brit. J. Radiol.* 1951, Jan., v. 24, No. 277, 38–40, 3 figs.

After a short account of toxoplasmosis with special reference to radiological findings, in which it is noted that intracranial opacities have been found in 85 per cent. of the cases of this disease recorded in the literature, two cases are reported :—

The first case was that of a newborn infant, weight 6 lb. On the day of birth the infant had a generalized macular rash, was jaundiced, and had a large liver and spleen ; an X-ray of the skull showed multiple discrete foci of calcification arranged in linear streaks and scattered throughout the fronto-parietal portions of the cerebral hemispheres and in the region of the basal ganglia. The rash persisted for 10 days.

The diagnosis of toxoplasmosis was "confirmed" by the complement-fixation test (positive at 1 in 30) and Sabin's dye test. Both tests were positive in the mother [titres not given].

Later, at 10 weeks, the child developed hydrocephalus.

The second case was that of a premature infant of 4 lb. 12 oz. At birth the infant was covered with a petechial rash, and was oedematous, and the liver and spleen were enlarged. The infant died within 15 minutes of birth.

X-ray of the skull carried out shortly after death showed the presence of scattered opacities throughout the cerebral hemispheres and in this case the curvilinear opacities were particularly well seen in the mid-parietal and occipital regions.

Toxoplasma were found in the heart muscles. The mother's serum gave a positive complement-fixation test for toxoplasmosis (1 in 30). *L. E. Napier*

Debré, R., Bertrand, I., Bargeton, E. & Mozziconacci, P. Toxoplasmose congénitale du nouveau-né. Observation anatomo-clinique. [**A Case of Congenital Toxoplasmosis. Anatomo-Clinical Study.**] *Bull. et Mém. Soc. Méd. Hôpit. de Paris.* 1951, Nos. 13/14, 553–9, 2 figs.

This paper describes a case of congenital toxoplasmosis occurring in a male child who was born with difficulty after a normal pregnancy and showed from the beginning hydrocephalus (38 cm.) and haemorrhagic meningitis. Ventricular puncture demonstrated a xanthochromic cerebrospinal fluid with 16 white cells per cmm., rising to 270 cells 13 days later. The retina showed a pigmented plaque of chorio-retinitis, and radiography of the head revealed about 10 small calcifications seen best near the widely open anterior fontanelle. The course of the disease was chequered, with febrile crises accompanied by convulsions, in one of which the child died 17 days after birth. The sera of mother (otherwise healthy) and child both gave positive neutralization reactions to *Toxoplasma*.

At autopsy, the surface of the brain was found flattened and covered by thickened haemorrhagic meninges ; on section, the lateral ventricles were seen to be enormously dilated and lined with a thick yellowish membrane, the brain substance was much reduced and contained areas of calcification. Histologically, the characteristic lesions of toxoplasmosis were observed—in particular, miliary glial granulomata near the necrotic areas and perivascular infiltration of lymphocytes, plasma cells and histiocytes. The organisms were seen either free or in pseudo-cysts (up to 16 μ in width) near the lesions. The parasites themselves were 2 to 3 μ long, rather scanty in number—they appeared to disintegrate rapidly in the pseudo-cysts which finally became calcified. No organisms were found elsewhere in the body. *P. C. C. Garnham*

Lelong, M., Le Tan Vinh, R. J., Desmonts & Dupré-Bouteloup, Lise. Un cas de toxoplasmose du nouveau-né. Isolement du parasite et étude anatomo-histologique. [**A Case of Congenital Toxoplasmosis. Isolation of the Parasite and Anatomo-Histological Study.**] *Bull. et Mém. Soc. Méd. Hôpit. de Paris.* 1951, Nos. 13/14, 541–53, 7 figs.

A case of congenital toxoplasmosis is reported in masterly detail. A male child was born at term after a pregnancy threatened in the eighth month with miscarriage. Breathing was established with a little difficulty, but it was not until eleven days after birth that abnormalities were noticed. Hydrocephalus (38·5 cm. perimeter) was then seen, also nystagmus and somnolence. The child was undernourished, with a sub-normal temperature ; his condition rapidly

deteriorated and he died on the sixteenth day of life. A lumbar puncture had revealed a yellowish fluid, under pressure, and containing 12·6 lymphocytes per cmm.

A post-mortem examination was made. The surface of the brain showed whitish streaks 2–3 cm. in width, with a gelatinous thickening of the pia mater, and visible through this at irregular intervals small nodules 2–3 mm. in diameter. Distension of the ventricles was marked, each ventricle was lined with a thick white membrane and contained a xanthochromic fluid. The white streaks seen on the surface of the organ formed part of an extensive internal necrosis, containing calcified elements. Smears of the brain showed typical toxoplasms (4–6 μ long) intra- or extra-cellular in position. In sections stained with Giemsa or Hortega, the organisms were smaller, and with the latter the nucleus was seen to contain 6–8 intensely black granules surrounded by a clear halo in the cytoplasm. Emulsions of brain were inoculated into white mice and the strain was isolated ; it is now in the 33rd passage. Immunity tests revealed no difference between this strain and the RH of Sabin or the Lyde strain.

The histological lesions were typical of such infections. In addition, the presence of eosinophilic monocytes in the cellular infiltrate was noted, and also a condition of endarteritis with toxoplasms present both in the capillary lining and lumen. Characteristic lesions were found in the retina. Organisms elsewhere were found only in the muscle cells of the heart which they distended to form an appearance resembling a " bag of marbles ".

The exact origin of this case is not mentioned, but it was presumably in the Paris region. The mother appeared perfectly healthy and had never suffered from a serious illness—nevertheless, the dye test of her serum was positive up to a dilution of 1 in 1,600. The authors stress that serological reactions alone are insufficient in our present state of knowledge to sustain a diagnosis of the infection, and that the only solid criterion is the discovery of the organism. The paper is illustrated by 7 photomicrographs. *P. C. C. Garnham*

JELLIFFE, D. B. **Congenital Toxoplasmosis in an African Child.** *Arch. Dis. in Childhood.* 1951, June, v. 26, No. 127, 258–60, 4 figs.

A presumptive case of toxoplasmosis is described in an African child living in Ibadan, Nigeria. The child was three years old and had had an uneventful life. The birth was said to have been normal, and the mother gave no history of any special illness. The child walked at 1½ years, talked at 2 and on examination showed no signs of deficiency. The head was greatly enlarged, measuring 60 cm. in circumference with a widely open anterior fontanelle. Radiography showed the presence of scattered areas of pin-point calcification in small clusters. The heart was enlarged, but the eye showed no abnormalities. Serological tests were performed by Dr. Sven Gard of Stockholm and Dr. Macdonald of Liverpool. The dye test was positive 1 : 50 in the mother and 1 : 2,000 in the child ; the complement fixation test also gave positive results. The paper is illustrated by 3 photographs and a diagram of the radiograph of the skull. *P. C. C. Garnham*

KLENERMAN, Pauline. **Congenital Toxoplasmosis.** *South African Med. J.* 1951, Apr. 21, v. 25, No. 16, 273–4.

An African male child, aged 1 month 10 days, was admitted to hospital in Durban and died in two days. The child was marasmic, afebrile, showed a bulging anterior fontanelle, and was semi-comatose. The cerebrospinal fluid

was under pressure ; the fluid appeared clear, but there was a marked increase of globulin and a " fair number " of lymphocytes were present. No micro-organisms were demonstrated. A diagnosis of tuberculous meningitis was made. Neither X-ray of the skull nor retinoscopy was carried out.

At autospy, hydrocephalus and scattered areas of necrosis in the brain were found. There were no tuberculous lesions of the meninges or lung. " Section of the brain showed considerable necrosis, especially around the fourth and lateral ventricles with a surrounding early gliosis and infiltration of eosinophils and phagocytic cells. There were numerous other scattered foci of microglia. Many of these swollen phagocytic cells contained groups of coccoid bodies as seen in toxoplasmosis.

" There was widespread oedema, perivascular cuffing, petechial haemorrhage and diffuse astrocytic proliferation in the wall of the ventricles. Focal areas of necrosis, with surrounding phagocytic cells, were present. In these areas were seen ovoid cells about 15 to 20 μ in diameter packed with minute basophilic ovoid-shaped bodies about 2 μ in diameter as seen in toxoplasmosis. No extracellular organisms were seen and no calcification was present in sections from different parts of the brain. No lesions were found in other organs."

Dr. Albert B. SABIN, to whom material was sent, identified the bodies as *Toxoplasma*.

The mother's serum gave the dye test at a titre of 1 in 1,024 and the father's serum at 1 in 64 (the highest limit of normality). Dr. Sabin agreed that this was a case of congenital toxoplasmosis. *L. E. Napier*

STRÖM, J. **Toxoplasmosis due to Laboratory Infection in Two Adults.** *Acta Med. Scandinavica.* 1951, Mar. 17, v. 139, No. 3, 244–52, 1 fig. [12 refs.]

The subject of congenital, childhood and adult toxoplasmosis, since Janku's first reported case in an infant in 1923, is reviewed summarily. The author then reports two cases of toxoplasmosis in which the infection was acquired in the laboratory.

A woman, aged 22 years, who was employed in a laboratory where work with *Toxoplasma* had been done for 18 days, developed an acute febrile attack with lymphadenitis, conjunctivitis, chills and headache. A reddish-brown maculo-papular rash on the trunk appeared on the third day, increased during the next few days, lasted two weeks and was followed by protracted desquamation. The fever was high (40·5°C.) remittent for four days ; then it fell but remained as a low irregular fever for about 14 days. There was a moderate leucopenia (3,600 per cmm.) for the first two weeks. The sedimentation rate was 14 mm. at first, but rose to 53 mm. after two weeks. Lumbar puncture showed evidence of mild meningeal irritation with an increase in protein and cells ; this persisted for three weeks.

After two months the patient was still complaining of severe headaches, mental exhaustion, marked inability to concentrate, and frequent attacks like petit-mal, which indicated encephalitic involvement. These continued, decreasing in severity, for six months. The electroencephalogram was negative on three occasions. Mild cardiac symptoms were present and had not altogether subsided after six months. The electrocardiogram at three weeks showed definite signs of myocarditis.

There were no retinal changes and no cerebral calcifications.

Toxoplasma were not demonstrated. Skin tests were faintly positive after a month. The dye test was at first negative, but after a fortnight it was positive at 1 in 250 and a week later at 1 in 4,000. After five months the titre began to fall.

The second patient was a woman of the same age, similarly employed, who pricked her finger with a needle contaminated with *Toxoplasma*. Three days later she developed fever for one day, slight local adenitis and lymphangites which disappeared in a week. The dye test was negative for two weeks, but later became positive (1 in 2,000). She had no other signs or symptoms.

L. E. Napier

PILLAT, A. Histologische Befunde am Auge bei konnataler Toxoplasmose. [**Histological Findings in the Eyes in Congenital Toxoplasmosis.**] *Wien klin. Woch.* 1951, Sept. 7, v. 63, Nos. 35/36, 628–31, 4 figs. [16 refs.]

An account of clinical material and discussion.

CHRISTIANSEN, M. & SIIM, J. C. **Toxoplasmosis in Hares in Denmark. Serological Identity of Human and Hare Strains of Toxoplasma.** *Lancet.* 1951, June 2, 1201–3, 1 fig.

Between 1935 and 1950 the authors have examined 2,812 wild hares (*Lepus europaeus*) in Denmark, among which 264 (9·4 per cent.) showed symptoms of toxoplasmosis. Autopsy of the infected animals revealed characteristic pathological changes in the spleen, liver, mesenteric lymph-nodes, and lungs. The well-nourished state of the animals suggested an acute course of the disease. Toxoplasms were found in large numbers in the organs, especially the spleen and brain.

This survey showed that in Denmark the height of the incidence of hare toxoplasmosis is during the cold season (January to March). There was no evidence of enzootic infections in any locality with a high hare population, only a few infected animals having been encountered in the same herd.

The hare strain was isolated by inoculating mice with a spleen suspension containing the parasites. Mice infected intraperitoneally or intracerebrally died in 4–6 days ; rabbits inoculated from mice died in 8–10 days, and hares infected by various routes died in 7–9 days with pathological lesions indistinguishable from those produced in these animals with a human strain and those occurring in spontaneous infections.

Serological tests carried out with 24 infected hares reacted positively with titres from 250 to 6,250 in the case of the dye test, and from 32 to 1,024 in the case of complement-fixation. Cross-neutralization and cross-complement-fixation tests with the human and hare strains of *Toxoplasma* showed that they were serologically identical.

It is concluded that people handling recently dead hares with a toxoplasmic infection run the risk of becoming infected. Since the toxoplasms survive only for a short time in infected tissues, it is advisable—especially for pregnant women—to handle hare carcasses only after they have hung for several days. It is pointed out that, since house dogs in Copenhagen gave significant positive serological reactions for toxoplasmosis, they might also play an important rôle in the spread of this disease to man.

C. A. Hoare

RUCHMAN, I. & FOWLER, J. C. **Localization and Persistence of *Toxoplasma* in Tissues of Experimentally Infected White Rats.** *Proc. Soc. Exper. Biol. & Med.* 1951, Apr., v. 76, No. 4, 793–6.

For this study a human strain of *Toxoplasma*, maintained in mice, was used. Rats were infected by intraperitoneal inoculation of a 10 per cent. suspension (in saline) of ground brain tissue from infected mice. At various intervals the rats were killed and the presence of toxoplasms was established by intraperitoneal subinoculation of 1 cc. of their blood or tissue suspensions

into groups of mice. The results were checked by microscopical examination of stained smears of mouse brains.

Toxoplasms were found in the circulating blood of rats in 4 to 72 hours, reaching maximum numbers by the 4th and 5th days, but after the 6th day and up to the 16th day they occurred only sporadically. Dilutions of 1 : 100 and, on one occasion, 1 : 1,000 produced infections in mice.

Subinoculation of tissue suspensions revealed the presence of parasites after 4 hours, in the liver, spleen and lungs ; they afterwards occurred more regularly for 7 days in the liver, 9 days in the spleen and 11 days in the lungs, but later they were only occasionally present for 10 weeks in the liver and lungs, and for 2 weeks in the spleen. In the brain, the toxoplasms were found at 4 days, after which they could be regularly detected for 2 years. The prolonged survival of the parasites in the brain of rats provided a method for maintaining the strain in these animals with long intervals between passages. Thus, 4 consecutive passages through rats were made over a period of 32 months, during which the parasites in the brain not only survived but had multiplied. The strain is now kept in rats, and when the parasites are required, mice are inoculated with brain tissue from an infected rat.

Antibodies appear in the blood of infected rats after the 1st week following inoculation, *i.e.*, during the decline of parasitaemia. The rats produce complement-fixing and cytoplasm-modifying antibodies after one week, and neutralizing antibodies at the end of a fortnight.

The rat can thus be used for various laboratory studies of toxoplasmosis : to demonstrate parasitaemia or any of the serological tests in use.

C. A. Hoare

ENTOMOLOGY AND INSECTICIDES : GENERAL

[Papers on the toxic effects of insecticides in man are abstracted in the *Bulletin of Hygiene* under the general heading of Occupational Hygiene and Toxicology.]

MENON, M. A. U. **On certain Little-Known External Characters of Adult Mosquitoes and their Taxonomic Significance.** *Proc. Roy. Entom. Soc. of London.* Ser. B. 1951, June 15, v. 20, Pts. 5/6, 63–71, 3 figs. [10 refs.]

CHOW, C. Y. & MATTINGLY, P. F. **The Male Genitalia and Early Stages of** *Aëdes* (*Finlaya*) *albocinctus* **Barraud and** *Aëdes* (*Finlaya*) *albotaeniatus* **var.** *mikiranus* **Edwards with some Notes on Related Species.** *Proc. Roy. Entom. Soc. of London.* Ser. B. 1951, Aug. 15, v. 20, Pts. 7/8, 80–90, 6 figs.

REEVES, W. C. **Field Studies on Carbon Dioxide as a possible Host Simulant to Mosquitoes.** *Proc. Soc. Exper. Biol. & Med.* 1951, May, v. 77, No. 1, 64–6. [11 refs.]

The author reminds us that a number of substances, carbon dioxide among them, are known to be attractive to female mosquitoes. He here reports preliminary studies made in the field in California. He used two "stable traps" [a type of cage with re-entrant slits in which one may put an animal bait]. One trap was baited with some pounds of dry-ice [solid CO_2], enough to last for a whole night, the other with a calf or with several chickens. On a considerable number of trials, the trap with CO_2 caught more female

mosquitoes than that with either type of live bait. This was shown by *Culex tarsalis* and *quinquefasciatus* [*fatigans*] and *Aëdes nigromaculis*. Unbaited traps caught almost no mosquitoes.

As the author points out, the method promises well. CO_2 is a simple standard bait: for some work (*e.g.*, search for viruses) one might prefer mosquitoes which had not been able to feed in the trap. It would certainly be better to avoid the lowering of temperature caused by the dry-ice, and to use some other source of carbon dioxide. *P. A. Buxton*

KETTLE, D. S. **The Spatial Distribution of** *Culicoides impunctatus* **Goet. under Woodland and Moorland Conditions and its Flight Range through Woodland.** *Bull. Entom. Res.* 1951, Aug., v. 42, Pt. 2, 239–91, 13 text figs. & 7 figs. on 2 pls.

KIRCHBERG, E. Untersuchungen über die Fliegenfauna menschlicher Fäkalien. [**Study of the Fly Fauna of Human Faeces.**] *Ztschr. f. Hyg. Zool.* 1951, May–June, v. 39, Nos. 5/6, 129–39, 2 figs.

Epidemics of diarrhoea and typhoid occurred in Berlin in 1945. As a result of war damage to the sanitation system, human faeces were at that time commonly found in the open throughout the city. An investigation was begun of the flies frequenting the stools. Traps, based on the principle of the Bruton trap, were used. The lower part consisted of a tray, with a central hole 20 cm. in diameter over which was inverted a large tea-strainer. The upper part of the trap consisted of a cage fitting firmly over the trap and provided with slots at its lower limits for the entry of flies. The roof of the cage was of sheet metal, protecting the cage and tray from the weather. Traps were set up with the stool within the arch of the tea strainer. Flies attracted to the faeces entered the cage through the slots and, for the convenience of handling the catches, were inactivated and killed by a DDT deposit on the inner surfaces of the trap. The traps were cleared daily after sunset. With the use of about eight traps during the summer a total of 2,346 flies were caught. The interesting species and their relative frequency were as follows: *Lucilia sericata* (53·5 per cent.); *Sarcophaga* spp. (12·7 per cent.); *Muscina stabulans* (12·1 per cent.); less than 1 per cent. of *Calliphora erythrocephala*, *Lucilia caesar* and related species, and a few other species; and less than 0·1 per cent. were *Musca domestica* (one female), *Fannia* sp., *Pollenia* sp. and some other flies. Female flies predominated, but considerable numbers of males of the three most frequent species also came to the traps.

The negligible catch of *Musca domestica* is discussed. Similar findings are reported from the United States of America and from Hungary. The author concludes that the rôle of the common house-fly as an agent in spreading human excremental diseases, at any rate in temperate regions, has been exaggerated. He attaches more importance to *Lucilia sericata*, which was found to visit the traps in large numbers and occurred also in houses, and markets in Berlin, where they fed or oviposited on fish, meat, and cheese. This species was also seen commonly on dog faeces in Berlin and the possibility of it serving as a carrier of toxoplasms and spirochaetes is mentioned briefly. It bred particularly in the bodies of rats and other animals in rubbish dumps in the city. There is some evidence to suggest that *Sarcophaga carnaria*, *S. haemorrhoidalis* and *Muscina stabulans*, which were also taken in appreciable numbers in the traps, may in addition frequent human foods (meat, fish, and cheese) sufficiently to act as carriers of disease organisms from human faeces.

D. S. Bertram

JETTMAR, H. M. & ANSCHAU, M. Versuche mit Culicideneiweiss-präzipitierenden Seren. [**Precipitin Sera Studies with Culicidae.**] *Arch. f. Hyg. u. Bakt.* 1951, v. 135, No. 1, 44–51.

The English summary appended to the paper is as follows :—

" Emulsions of numerous insects belonging to the order Diptera were tested by means of antisera of rabbits immunized by intraperitoneal inoculations with emulsions of culicine pupae. Following results were obtained :

" 1. The albumen of insects not belonging to the order of Diptera has never been precipitated by the culicine pupal antiserum.

" 2. Emulsions from *Musca domestica* showed only weak precipitation with a very low titre (not higher than 1 : 20).

" 3. From the examined insects belonging to the Subordo Nematocera the representatives of the families Dixidae, Corethridae, Simuliidae and Bibionidae showed high titres which in some cases approached to the titres of the specific antigen (culicine pupal emulsion).

" 4. Anophelines and Aedines showed precipitin titres which were identical to the specific antigen.

" 5. No difference could be observed between the larval and pupal emulsions of the identical species.

" 6. The albumen of insect predators fed upon Nematocera (not only Culicidae but Corethridae a.o.) gave a strong positive precipitin test. Even after starvation up to 7 days this test remained positive although the titre became low.

" 7. Our experiments and observations show that the application of the precipitin test to determine the presence of culicide mosquitoes ingested by insect predators in a certain environment can have only—if ever—a very limited practical significance."

JENSEN, J. A. & FAY, R. W. **Tagging of Adult House Flies and Flesh Flies with Radioactive Phosphorus.** *Amer. J. Trop. Med.* 1951, July, v. 31, No. 4, 523–30.

A method has been developed for marking flies with radioactive phosphorus, in order to study their movements in the field. The species chosen for experiments were *Musca domestica*, *Callitroga macellaria* and *Phaenicia pallescens*. These flies could be made radio-active by adding radio-active phosphate ($NaH_2P^{32}O_4$) solution to either the larval rearing medium or to milk on which adult flies fed. In the first method, however, only very low levels of activity were found in the adult unless the concentration of P^{32} in the medium was so high (0·18 microcuries per gramme) as to have adverse effects on larval growth.

By suitable experiments, the daily milk consumption of adult flies was found and the absence of repellent effect due to phosphate was ascertained. Based on these results, and assuming 40 per cent. absorption, and detection of 10 per cent. of the radio-activity, the concentration of P^{32} calculated to give a significant count on a Geiger-Muller counter at 2½ inches, was about one microcurie per millilitre.

Groups of twenty flies were offered 5 ml. of radio-active milk daily for 1, 2 or 3 days. It was found that uptake of P^{32} increased with longer feeding, more rapidly in females than in males. If the subsequent diet was milk, one day's feeding gave radio-activity lasting only 3 days, but with 2 or 3 days' feeding, detectable activity was present after 12 days. Uptake also increased with increase in concentration of P^{32} up to 4 microcuries per ml. but it is not known

whether this is harmful to the flies. The age of the fly, up to 6 days, did not significantly affect the uptake of P^{32}, but, at the age of 6 days, uptake was slightly less.

The decline of radio-activity depends on the phosphorus content of the food, being much more rapid, on account of phosphorus exchange, on a milk diet (rich in phosphorus) than on honey. This should be borne in mind in field work, and releases at too short intervals should be avoided, since the radio-active flies of successive batches might not be easily distinguished.

Trials showed that radio-active flies can be quickly detected in trap catches or on baits, the examination of dead flies from traps being the more accurate method. *A. J. P. Goodchild*

COLUZZI, A. & RAFFAELE, G. Indagini sull'azione residua del DDT e del Chlordane sulla mosca domestica e sugli anofeli. [**Study of the Residual Action of DDT and Chlordane on House-flies and Anophelines.**] *Riv. di Malariologia.* 1951, June, v. 30, No. 3, 113–36, 18 figs. English summary.

The investigations comprised both laboratory tests and field observations. In the laboratory tests, batches of house-flies were exposed to residues of DDT and chlordane in small treated cages, for different intervals (5 to 30 minutes), and then kept in clean cages for 24 hours before mortality counts. The treated cages were coated with plaster and were hinged so that they could be opened for treatment. This was given with a sprayer applying kerosene solutions to give 2 gm. of insecticide per sq. metre. Throughout, the initial and residual insecticidal action of the chlordane was somewhat superior to that of the DDT.

The same type of laboratory test was used to compare the resistance of flies from areas regularly sprayed with DDT, or DDT plus chlordane, for 3 years, with localities where no spraying had been done. The time for complete mortality, by DDT, of flies from 3 untreated zones was not appreciably different from the time for flies from treated zones. [This differs from the observations of most other investigators. Perhaps the "previous spraying" was not sufficiently widespread or thorough to select out the resistant strains of flies.]

In the field, the effectiveness of treatments were judged by periodic fly counts at a number of localities. A comparison of data for DDT-treated houses with those for untreated ones, shows only a slight improvement ; but after chlordane treatment, there was a sharp fall in numbers maintained for several months. Nevertheless, the authors are not convinced that the insecticide was the sole cause of the diminution in flies.

The effectiveness of DDT against domestic *Anopheles* was observed to be much more satisfactory. One treatment kept the numbers insignificantly low for 14 months. *J. R. Busvine*

MOSEBACH, Erna. Unterschiedliche Empfindlichkeit von Männchen und Weibchen der Stubenfliege. [**Susceptibility (to DDT and BHC) of Male and Female House-flies.**] *Ztschr. f. Hyg. Zool.* 1951, May–June, v. 39, Nos. 5/6, 139–45, 3 figs.

An account is given of some experiments in which mixed populations of both sexes of *Musca domestica* were exposed to DDT or BHC [concentrations not stated]. The results are tabulated. Male flies were knocked down earlier than most of the female flies ; in the case of a light dose of DDT, all but one of 10 females recovered, but most males did not. Thus, in these experiments, the original population of about equal numbers [usually] of males and females becomes one in which females predominated. The author goes on to state that

a similar selection of females occurs in laboratory cultures or in nature when some adverse condition causes earlier deaths among the males. Normally, it is stated, the sexes are equal in nature, and a table is given showing catches of almost equal numbers of each sex on five separate occasions. From all this, it is concluded that the sex-ratio of a house-fly population indicates whether the conditions to which it has been subject in the past have been favourable or not. There is a general suggestion that this should be taken into account when assessing the effects of adult fly control [presumably by insecticides]. In a population of about equal numbers of both sexes, the effects of control would be more apparent, owing to the greater susceptibility of the males, than if the original population was, by reason of earlier adverse circumstances [insecticidal or natural], mainly of the more resistant females.

It is also suggested in the light of these observations, that more exacting tests with more sensitive results would be possible in experiments with [presumably] insecticides if female flies only were used as the test insect.

D. S. Bertram

ASCHER, K. R. S., REUTER, S. & LEVINSON, Z. **The Insecticidal Properties of DDT as related to its Physical State in Residue. Laboratory Experiments on Glass-Surfaces.** *Advances in Insecticide-Research.* Jerusalem. 1951, Jan., No. 1, 18 pp., 2 figs.

A spray of a 5 per cent. solution of technical-grade DDT used in houses in Israel gave an adequate control of house-flies for only the first few days. Since there was no chemical or quantitative change in the DDT deposit it seemed probable that loss of effectiveness was due to a change in the physical state (probably crystallization) of the insecticide.

The knock-down of DDT-resistant flies (*Musca domestica* var. *vicina*) when exposed to glass surfaces treated with DDT in kerosene, was compared with that of flies exposed to similar surfaces treated with a formulation of DDT in kerosene and benzyl benzoate which prevented crystallization of the DDT. The best results were obtained with p-p′ DDT and benzyl benzoate in the ratio of 2 : 5.

It has also been noticed that technical-grade DDT frequently has a slightly longer toxic effect to the house-fly than p-p′ DDT. Technical DDT was separated into the two fractions p-p′ DDT, and the "rest" by treatment with ethyl alcohol. The "rest" fraction gave better results than either p-p′ DDT or technical-grade DDT alone when tested against house-flies. A formulation containing 10 per cent. p-p′ DDT and 90 per cent. "rest", or 5 per cent. p-p′ DDT and 95 per cent. "rest", when applied to a glass as 4 per cent. solutions in standard kerosene, gave a quicker knock-down and remained effective for a longer time than any other formulation. *C. Mary Harrison*

ESTHER, H. Über das Auftreten "DDT-resistenter" Fliegen (*Musca domestica* L.) im Jahre 1950. [**Appearance of DDT Resistant House-flies in Germany in 1950.**] *Deut Gesundheitswesen.* 1951, Aug. 23, v. 6, No. 34, 967–70, 2 figs.

GERSDORFF, W. A. & MITLIN, N. **Joint Toxic Action against House Flies in Mixtures of Parathion and its Methyl Homolog.** *J. Econom. Entom.* 1951, Aug., v. 44, No. 4, 474–6.

"The relative toxicity to adult house flies, *Musca domestica* L., of parathion, its methyl homolog, and three mixtures of these compounds was determined by the turntable method.

"The methyl homolog was 68 per cent as toxic as parathion. The mixtures of parathion and its methyl homolog (1 : 4, 1 : 1, and 4 : 1) were, respectively, 72, 88, and 91 per cent as toxic as parathion.

" The action of the compounds when applied jointly was identified as similar action since one of them can be substituted for the other in a constant ratio without altering the toxicity of the mixture."

OSIMANI, J. J. Brote epidémico de miasis humana forunculosa por *Dermatobia hominis* Linn. [**Outbreak of Myiasis in Man, due to** *Dermatobia hominis*.] *Arch. Uruguayos de Med., Cirug. y Especialidades.* 1951, Mar., v. 38, No. 3, 119–25.

At the end of December 1950, 7 persons went to the Fortress of Saint Teresa, in the Department of Rocha, some 308 kilometres from Montevideo and stayed there for 11 days. Six of them developed a pustular myiasis due to *Dermatobia hominis*. All six of them wore little clothing and that of the lightest ; men and women both wore shorts and the men thin shirts without sleeves. The only one to escape was a man of 55 who was clothed in a jacket and long trousers.

The insect is believed to have been introduced with Venezuela cattle. Human infestation is not uncommon in the country, but is greatest in the Canelones, Maldonado and Rocha zones. In prevention, it is almost impossible to deal with the adult fly, but application to the animals of a suspension of derris, 1 in 10, with water 9 parts and soap emulsion 1 part, applied for 3 days every fortnight is successful. For man, the best repellent seems to be dimethyl phthalate. It is feared that unless the infection is checked, knowledge of it will hinder the tourist trade. *H. Harold Scott*

LEESON, H. S. **The Recorded Distribution of the Tick** *Rhipicephalus sanguineus* **(Latreille).** *Bull. Entom. Res.* 1951, July, v. 42, Pt. 1, 123–4, 1 fig.

In 1941, BUXTON set out the world distribution of certain fleas in 5 maps and an explanatory text [this *Bulletin*, 1942, v. 39, 306].

The present author has accumulated information on the same lines in relation to the tick *Rhipicephalus sanguineus* and has brought the search of the literature up to January, 1949.

R. sanguineus was first recorded by LATREILLE from France in 1806 as *Ixodes sanguineus*. Thereafter, it was rarely mentioned (and not always by that name) until NEUMANN's monograph in 1911. Throughout the intervening 100 years or so it was recorded from East Africa as *R. stigmaticus*, but later the distribution of this " characteristically African " tick was found to be extended to Italy, Asia, Central and South America. The first reference to the tick in North America was made by BANKS in 1908, when he referred to it as *R. texanus* from Texas and New Mexico. Thereafter the tick has been recorded with increasing frequency from so many countries that COOLEY in 1946 called it the most widely distributed tick in the world.

The basis of this statement is well illustrated in the map presented by the present author, in which the localities where *R. sanguineus* has been reported are shown in black, and it is clear that the range of distribution is considerable.

The tick has been found on many animals, but the dog is its usual and preferential host. *H. J. O'D. Burke-Gaffney*

ERRATUM

In the abstract of the paper by ZUMPT & GRAF in this *Bulletin*, 1951, v. 48, 295, the abstracter suggested that the mite *Pneumonyssus* occasionally causes pulmonary infections in man. He now writes that this suggestion is mistaken. It is, of course, well known that it infests the respiratory passages of monkeys.

SIMMONS, S. W. & UPHOLT, W. M. Los insecticidas en el control de las enfermedades. Revisión de la literatura. [**Insecticides in the Control of Disease. Review of the Literature.**] *Bol. Oficina Sanitaria Panamericana.* 1951, July, v. 31, No. 1, 1–21. [104 refs.]

This is a Spanish translation of the paper abstracted in this *Bulletin*, 1951, v. 48, 782.

DOWNS, W. G., BORDAS, E. & NAVARRO, L. Duración de la efectividad de depositos residuales de DDT en superficies de adobe. [**Duration of Effectiveness of Deposits of DDT on Adobe Surfaces.**] *Medicina.* Mexico. 1951, June 10, v. 31, No. 629, 215–20, 1 fig. [18 refs.]

The authors comment on the wide differences, in residual action of DDT against mosquitoes, observed by various investigators. This may be partly due to differences in susceptibility of the mosquitoes concerned ; but sometimes the DDT appears to be more persistent in some localities than in others of the same country. This is very evident in Mexico, as shown by tests in which mosquitoes (*Anopheles aztecus* and *A. albimanus*) were exposed for short periods, against the walls of treated houses, at various intervals after treatment. Six houses were treated in each of four localities, being sprayed with DDT suspension at 2 gm. DDT per sq. metre. The results within each group were consistent. In one locality the walls of the houses were highly insecticidal three years after treatment ; in the second group the effect began to fail in the second year ; in the last two groups the insecticidal action fell off in the first year.

These houses were constructed with adobe walls, made from local materials. It was suspected that some types might more readily decompose DDT than others, and this was confirmed by chemical tests in which the powdered adobe was mixed with DDT and exposed to a high temperature. There was very good inverse correlation between persistence of insecticidal effect and the rapidity of decomposition. A careful analysis of the adobe materials revealed that those on which the DDT was unstable contained considerable amounts of iron and aluminium, which are known to catalyse the decomposition of DDT. *J. R. Busvine*

KEMP, H. & DE AGUIAR, Hilda A. Método turbidimétrico para a determinação de pequenas quantidades de DDT. [**Turbidimetric Method of Determining Small Quantities of DDT.**] *Rev. Brasileira Malariologia.* Rio de Janeiro. 1950, Oct., v. 2, No. 4, 318–25, 2 figs. English summary (5 lines).

There are several colorimetric methods of estimating DDT in residues on walls, but they all are liable to suffer from interference, if the walls are coloured or if the DDT is applied with an emulsifier which reacts chemically to give a colour.

The authors have developed a turbidimetric method which satisfactorily measures 1 to 5 milligrammes of DDT. It depends on hydrolysing the DDT with alcoholic KOH, adding silver nitrate and estimating the precipitate with a spectrophotometer. Preliminary tests showed that (i) the precipitate was fully developed in about half an hour, (ii) that maximum absorption of light occurred at 4,000 Å, and (iii) that the density of alcohol used for dilution was important.

Method.—Scrape the residue from the wall and put it in a tube with 5 ml. of 0·2N KOH (in 96 per cent. alcohol). Heat on a water bath at 80°C. for 5 minutes, then allow the powder to settle for ½ hour. Pipette 2 ml. clear liquid into a "pyrex" tube, dilute with 10 ml. alcohol (d=0·896), add 0·3 ml.

(5 drops) of 4N nitric acid and shake. Measure the absorption on the spectrophotometer at 4,000Å. Add 1 ml. (15 drops) of 0·1N silver nitrate, shake and allow to rest for half hour before measuring the absorption again. The difference in the two measurements (due to precipitate) is compared with a calibration curve previously obtained with equal portions of an alcoholic solution of DDT. *J. R. Busvine*

HOGAN, T. W. **The Ground Recovery and Drop Spectra of Sprays dispersed from Two Types of Aircraft.** *Australian J. Agric. Res.* 1951, July, v. 2, No. 3, 302–21, 12 text figs. & 2 figs. on 1 pl.

FALES, J. H., NELSON, R. H., FULTON, R. A. & BODENSTEIN, O. F. **Tests with Two Insecticidal Aerosols for Use on Aircraft.** *J. Econom. Entom.* 1951, Aug., v. 44, No. 4, 621–2.

Since 1942, insecticidal aerosols have been used for the control of insects of public health importance on aircraft entering the United States. Early in 1948, after many formulae had been tested, the Bureau of Entomology and Plant Quarantine recommended a formula designated as "G 382", and this was officially approved by the U.S. Public Health service in 1948. [The experimental work on which this decision was based does not seem to have been published.] Certain users objected to the cyclohexanone employed in this formula, and a second ("G 651") was developed, in which this solvent was partly replaced by methylated naphthalenes. The two formulae are as follows :—

Composition (per cent.)	G 382	G 651
Pyrethrum extract (20%)	5	6
DDT	3	2
Cyclohexanone	5	2
Methylated naphthalenes	—	8
Lubricating oil	2	—
Percentage non-volatile constituents ...	15	16
Particle size (microns) MMD	14	14
Particle size (microns) largest	45	40

This paper reports comparative tests of the two aerosols in a Peet-Grayd chamber, in which flies and two species of mosquito were apparently given 15-minute exposures. The two formulae are clearly very similar in effectiveness for knock-down and kill ; but it is not certain how far this laboratory test can be regarded as a guide to practical conditions. The U.S. Public Health requirements state that these aerosols should be used at 5 gm. per 1,000 cu. ft. with a subsequent 3-minute closure.

[Two American aerosol bombs filled with the G 651 formula were tested in England in actual aircraft in 1950. See TEW, DAVID and BUSVINE, this *Bulletin*, 1951, v. 48, 589.] *J. R. Busvine*

FALES, J. H., NELSON, R. H., FULTON, R. A. & BODENSTEIN, O. F. **Insecticidal Effectiveness of Sprays and Aerosols containing Allethrin.** *J. Econom. Entom.* 1951, Feb., v. 44, No. 1, 23–8.

Experiments are described to determine the effect of the completely synthetic allyl homologue of cinerin I, known as allethrin, when used in sprays and aerosols

against house-flies, mosquitoes (*Aëdes aegypti* and *Anopheles quadrimaculatus*), and cockroaches (*Periplaneta americana*). All tests were made in a Peet-Grady chamber. Free-flying flies and mosquitoes were used, mortality counts being made 24 hours after exposure. Cockroaches were exposed for 30 minutes and mortality counts were made up to 5 days after.

Direct comparison was made between the synthetic ester and natural pyrethrins in sprays containing 1 mgm./ml. of deodourized kerosene, and aerosols containing 1 per cent. Equal mortalities were obtained in house-flies for the two materials by both methods, but the synthetic ester in aerosols gave slower initial knockdown. Comparative tests with kerosene sprays against the two species of mosquitoes at 0·2 mgm./ml. showed the synthetic ester to be less effective, and *Anopheles* to be more resistant than *Aëdes*. The synthetic ester at 1 mgm./ml. gave good results with both species. Aerosols containing 1 per cent. synthetic ester tested on cockroaches at 35 gm./1,000 c.ft. were found to be inferior to similar aerosols containing natural pyrethrins, although these were not very effective at the dose used. The distilled synthetic ester, when tested against houseflies in kerosene sprays at 1 mgm./ml., proved no better than an undistilled sample. A freshly made aerosol containing the latter, when compared with one stored for 4 months at room temperature, proved to be of equal value. The same aerosol was less effective against DDT-resistant flies than against the normal laboratory strain.

Comparison was made between the synthetic ester and pyrethrins in practical aerosol formulations. In high pressure aerosols, piperonyl butoxide, n-propyl isome and sesame oil extractions all showed synergism with the synthetic ester, although a marked recovery of affected flies occurred with the last. In a U.S. Public Health Service formula containing DDT, the synthetic ester and pyrethrins proved equally effective to both house-flies and mosquitoes. The synthetic ester, when substituted in the Tentative Official Test Aerosol of the Chemical Specialities Manufacturers' Association, gave kills equal to the pyrethrin formula and higher knockdown effects with house-flies. Three low-pressure aerosols containing DDT and a synergist were tested. One containing n-propyl isome acted similarly with the synthetic and natural pyrethrins, while Van Dyke 264 gave a higher kill, and piperonyl butoxide equal mortality and lower knockdown with the synthetic ester than with natural pyrethrins.

Ruth Nash

BOOK REVIEWS

CRAIG, Charles Franklin [M.D., etc.] & FAUST, Ernest Carroll [M.A., Ph.D.]. **Clinical Parasitology.** With a chapter on "Control of Medically Important Arthropods" by Albert MILLER [B.S., M.S., Ph.D.]. 5th Edition, thoroughly revised. 1032 pp., 326 engravings & 6 coloured pls. 1951. London: Henry Kimpton, 25, Bloomsbury Way, W.C.1. [84*s*.]

Medical parasitology made large advances during the second world war, particularly in the field of the cure and prevention of arthropod-borne diseases, while the widespread use of the new insecticides revolutionized our ideas about eradication of notorious infections such as malaria, plague and typhus. The last six years have seen the gradual perfection of techniques and the development of new policies. The time was therefore particularly appropriate for a new edition of this standard text-book on the subject, and the authors have

added new chapters embodying these advances while expanding the old ones. The long association of the two authors has continued to ensure a natural homogeneity to the book ; it is all the more sad to think that with the death of Charles Craig in 1950, future editions will be deprived of this advantage.

The new chapter by Albert Miller on the control of medically important arthropods begins with some fundamental principles which are a *conditio sine qua non* in tropical hygiene. It is emphasized that control depends upon a sound knowledge of the biological foundation of the problem, while refinements of control depend upon various types of specialized knowledge from engineering to psychology. Control entails interference with arthropods in their quest for food or shelter, and the measures for attaining this end are classified under the usual heads. The chief part of this chapter deals with chemical control, which is divided into control by (*a*) contact and stomach poisons, (*b*) fumigants, and (*c*) repellents. The various chemicals are described with brief notes on formulae, physical properties, toxicity to man and animals, and mode of employment as insecticides, with dosages. The list is preceded by a summary indicating *inter alia* the insecticides of outstanding merit for each arthropod. It is doubtful if general agreement could be obtained today on such a subject, and not everyone would agree for instance that DDT is a better insecticide for ticks than benzene hexachloride.

In a book on parasitology, the very nature of the subject makes it impossible to avoid some duplication ; the host, the vector and methods of diagnosis almost inevitably have to be described separately, and yet they must also be considered together. In this book, information is sometimes repeated in the different sections, sometimes not ; in consequence, in spite of the good index, it may be difficult to know where to run a certain item to earth. For instance, xeno-diagnosis for Chagas's disease is described in an introductory chapter, is redescribed under *Trypanosoma cruzi* and yet is omitted in the special chapter dealing with technique. In the same way, biopsy of skin in the diagnosis of onchocerciasis is mentioned in one section but not in the special diagnosis chapter later.

The sectional arrangement of the book remains the same as in previous editions : Introductory Section, Protozoa, Helminths, Arthropods and Human Disease, Technical Appendix, and Literature. The sections on the protozoa and helminths make this book a classic of its kind ; the description of each infection is so clear that a picture comes to life at once, and the uniform arrangement into synonyms, history, distribution, morphology, life cycle, epidemiology, pathogenesis, pathology and symptomatology, diagnosis, treatment and control throughout these two sections makes it ideal for the student. Much the same classification is used in the section on Arthropods and Human Disease, where certain diseases caused by bacteria, rickettsiae, bartonellae, viruses and spirochaetes are described. The Technical Appendix is obviously derived from the storehouse of the practical experience of the authors themselves, and is invaluable. It is clear that practical men are writing when they state that " the differential diagnosis of parasitic amoebae constitutes the most difficult problem in clinical parasitology ". Information about each infection has been brought up to date and there are many references to the literature of 1950 as well as much unpublished data based on recent observations by the junior author.

It is possible to make many minor criticisms of this book ; probably the chief is that the emphasis is too much on American instead of on world-wide parasitology. This is shown by the frequent use of the phrase " in this country " presumably meaning the United States, and there is a tendency to stress the American origin of new work. It is strange to find the names of Yu and Ying associated with the provocative action of adrenaline in the diagnosis of chronic

malaria, instead of that of ASCOLI. The book contains many mistakes, either of spelling or proper names or obvious slips ; also some of greater importance. The posterior nuclear forms of trypanosomes are called the " crithidia stage " ; in describing malaria gametocyte development in the vertebrate host, it is stated that the gametocyte when fully grown is " liberated by the destruction of the corpuscle " and remains free in the blood " serum " ; sternal puncture is said to have practically replaced spleen puncture in the diagnosis of kala azar ; one coloured plate illustrates a so-called mature male gametocyte of *P. vivax* occupying less than half the cell, while another shows schizonts of *P. falciparum* filling the erythrocyte (the coloured plates of thick film preparations on the other hand are excellent in their exact portrayal of stained parasites) ; *Anopheles pretoriensis* is designated an important malaria carrier ; the breeding places of *Aëdes aegypti* are said to be only domestic ; enzootic plague is stated to exist in rodents especially in " forested areas ", etc.

No books of this sort can be perfect and the type of error mentioned above hardly detracts from its immense usefulness. It differs from the usual text book on tropical diseases in that, firstly, it deals with the whole world instead of being confined to the tropical, and secondly, the accent throughout is on the aetiological agent and vector rather than on the disease. It is the sort of book (plus a microscope) that one would like to be shipwrecked with on a desert island. *P. C. C. Garnham*

HEDIGER, H. [Direktor des Zoologischen Gartens Basel.] **Kleine Tropen-Zoologie.** [Compendium of Tropical Zoology.] *Acta Tropica.* Basle. 1948 Suppl. 1. 182 pp.

It is not at first sight easy to see what place in the world of medical literature this book is destined to occupy, or should occupy. To the reviewer it would appear to be eminently suitable for the educated adult who is interested in zoology, especially if he has travelled abroad. For the medical man practising in the tropics and looking for help on medical matters the information is scanty. The author's official position and his previous publications indicate, we think, his point of view and the object of the present work.

Professor Hediger is Director of the Basle Zoological Gardens and is also on the staff of the University and of the Swiss Tropical Institute.

In his introductory chapter he devotes considerable space to what he designates " Fanciful Tropical Zoology " (*Phantastische Tropen-Zoologie*). He points out how erroneous are many of the popular ideas on the subject as told in writings and displayed in pictures and in films, even travel and expedition films intended to be instructional and educational ; many of the tales and films of the tropics, for example, would lead one to think that a venomous snake or some beast of prey was lurking behind every alternate tree.

The author tells how almost every animal in the wild has its enemies but that man is the universal enemy from whom wild animals all escape by flight if they can and that it is largely in man's imagination that he thinks every animal in nature is a menace. Man-eating lions and tigers are not normal, but rare exceptions, which have in many instances become so in old age when they can no longer chase and bring down their more nimble prey, or they turn on man when he approaches too near their lairs and where animals are tending their young, or when wounded and cornered and no longer able to escape by flight.

After the introduction comes a chapter on General Tropical Zoology, the distribution and numbers of the various zoological families and genera, mammals, reptiles, amphibia, butterflies, etc. in different countries. Section C, Special Tropical Zoology, occupies four-fifths of the whole. This section itself is

subdivided into : (*a*) Tropical wild animals, apes, elephants, ungulates and beasts of prey of the cat family, and also bears and birds, in particular vultures. (*b*) The domestic animals are next considered : the water-buffalo, zebu, sheep and goat, camel and, of birds, the ostrich for ostrich farming. (*c*) The third and largest subsection deals with poisonous animals of the tropics, rather from the anthropocentric than the biological aspect. The author states that there is a difficulty in defining "poisonous" for general purposes ; thus, common salt may be a poison for amphibia ; fine hairs may penetrate the skin and set up inflammation like a chemical poison, acetylcholine and adrenaline, produced in the human body, can act as poisons. He gives Pawlowsky's division into four varieties : (i) The stinging and biting insects and animals—*Tabanidae*, *Stomoxys*, fleas, bees, wasps, mites, scorpions, spiders, snakes. (ii) Including those with skin glands secreting poison ; salamanders, toads, Triton. (iii) Toxic fish and molluscs—*Tetrodon*, *Schizothorax*, by spines or as food poisoning when the flesh is eaten. (iv) Parasites, protozoa and helminths. [A poor and unsatisfying classification.] In this section he reverts again to animals with natural weapons of defence and offence, teeth, claws, hooves, horns, to the electric shock of the electric eel, Torpedo, to the offensive odour of the skunk and, lastly, to the psychological weapons as erection of the hair of the wolf or the hood of the cobra to frighten away enemies.

The sources of the poison next come up for discussion, the spines of the Echinoderm, the skin secretion of certain fishes and amphibia, the poison of centipedes, and he discusses very briefly the existence of poison in some individual species, the source of it, the poison apparatus, the mode of transference of the poison and the mode of action. The various poisons and poisonous animals are dealt with almost entirely in general terms with occasional examples ; thus spiders, illustrated by *Latrodectus mactans*, scorpions, repeating only to demolish the age-old tale of their suicide when cornered ; of fish by *Muraenidae*, *Physalix* and *Trachina* ; lizards by *Hyla* and *Heloderma* ; snakes by cobra, mamba and *Lachesis*, pointing out the distinctions between the proteroglypha, opisthoglypha and the aglypha ; the constrictors and sea-snakes are mentioned for completion.

There is presented a list giving the measurements of some of the more important snakes. Twelve families of snakes have each a paragraph describing clearly and succinctly their characteristics. There is also a useful list of the dangerous snakes of tropical Africa, tropical Asia, and tropical America, with both the scientific and their common names. The treatment of snake-bite is summed up in the use of antivenene, in general terms (no dose is mentioned) and in first-aid measures, as by tourniquet, cautery, excision, potassium permanganate, and the use of protective clothing such as leggings, care in walking and the avoidance of night-travelling.

To sum up : We have here an authoritative book of great interest from the point of view of the zoologist, but readers who look for *medical* details and instruction on poisonous animals of the tropics will be disappointed ; it is only fair, however, to add that, presumably, it was not for them that the work was written. For those interested in animal life and particularly for those living near zoological gardens who can, therefore, verify what they read, this work will be very welcome. *H. Harold Scott*

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Am.	signifies	Amoebiasis and Intestinal Protozoal Infections.
Bart.	,,	Bartonellosis.
Bl.	,,	Blackwater Fever.
B.R.	,,	Book Review.
Chl.	,,	Cholera.
Def. Dis.	,,	Deficiency Diseases.
Den.	,,	Dengue and Allied Fevers.
Der.	,,	Dermatology and Fungus Diseases.
Ent.	,,	General Entomology and Insecticides.
Ep. Dropsy	,,	Epidemic Dropsy.
Haem.	,,	Haematology.
Heat Str.	,,	Heat Stroke and Allied Conditions.
Hel.	,,	Helminthiasis.
Lab.	,,	Laboratory Procedures.
Leish.	,,	Leishmaniasis.
Lep.	signifies	Leprosy.
Mal.	,,	Malaria.
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Pl.	,,	Plague.
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Reports, etc.	,,	Reports, Surveys and Miscellaneous Papers.
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Benard, P., with Jausion, Calop & Aboud, (837) (Heat Str.)

Benavides, I., with Gajardo Tobar, Apablaza, Uribe, Vargas, Cepeda, Rojas, Zeldis, Lucchini & De la Fuente, 381 (Hel.)
Benchimol, A. B., with Schlesinger, 1139 (Def. Dis.)
Benetazzo, B. & Gambini, G., 277 (Hel.)
——, with d'Ignazio, 353 (Typh.)
—— & Pansa, G., (476) (Hel.)
—— & Zama, F., 385 (Hel.)
Ben-Harel, S., 526 (Mal.)
Benitez Gautier, C., with Meyer, Suarez, Martinez & Suarez, 916 (Sp.)
Benitez Soto, L., with Gradwohl & Felsenfeld, 1054 (B.R.)
Benn, K. M., 580 (Vms.)
Bennett, H. J., with Hunter, Dillahunt, Faust, Modic, Connellan & Ingalls, 570 (Hel.)
Benson, J. F., with Bauer, Parker, Hall, Joslin, Hightower, Snyder, Venable & Woodward, 979 (Typh.)
——, with Powell, Snyder, Minor & Woodward, 979 (Typh.)
Bentégeat, with Fontan, Verger, Martin & Dubreuil, (367) (R.F.)
Benyesch, M., with Dreyfuss, 831 (Haem.)
Bequaert, J. C. & de Lucena, D. T., 743 (Hel.)
Béquignon, R. & Vialat, C., 982 (Rab.)
Berberian, D. A., Dennis, E. W. & Pipkin, C. A., 154 (Am.)
Béréni, J., 495 (Vms.), 510 (Mal.)
Berg, J. A. G. Ten, *see under* Ten Berg
Berge, C., 577 (Hel.)
Bergeret, C., 668 (Def. Dis.)
Bergouniou, J. L., 1141 (Def. Dis.)
Berke, Z., with Berkin, 48 (Hel.)
—— & Cilesiz, A., 457 (Rab.)
Berkin, T. & Berke, Z., 48 (Hel.)
Berks, G. & Goodwin, L. G., 556 (R.F.)
Berman, C., 83 (Misc. Dis.), 845 (B.R.)
Bernard, P., 444 (Mal.)
Bernet, 111 (Mal.)
Bernet, J., (89) (Ent.), 126, (609) (Mal.)
Berry, E. G., Nolan, M. O. & Oliver González, J., 53 (Hel.)
Bertagna, P., 1068 (Mal.)
Bertín, V., with Neghme, Donckaster, Silva, Artigas, Bunster, Muñoz T., Faúndes & Basso, 484 (Hel.)
——, with ——, Tagle, Silva & Artigas, 484 (Hel.)
Bertram, D. M., 328, 331 (Mal.)
Bertram, D. S., 60 (Hel.), 546 (Typh.)
Bertrand, I., with Debré, Bargeton & Mozziconacci, 1147 (Prot.)
Bertrand, J., 829 (Def. Dis.)
Bervoets, W., with Wanson & Courtois, 414 (Ent.)
Besse, P., with Chaussinand, (1001) (Lep.)
Bettolo, M., (247) (Typh.)
Beukema, W., 895 (Am.)
Beutnagel, J., with Zenner & Friederich, (671) (Der.)
Bezerra, F. P. G., Jr., with Penido & de Sousa, 235 (Mal.)
Bharucha, K. H., with Sokhey & Habbu, 256 (Chl.)
Bhattacharya, B., with Sen Gupta & Mallick, 594 (Lab.)
Bhende, Y. M., Purandare, N. M., Banker, D. D., Figueredo, N. & Desai, S. D., 452 (Leish.)
Bhima, S., with Holmes, Gee, Asea, Chagula, Evelia, Kasirye, Kiboneka, Lugazia, Majale, Mughairy, Ochieng, Gesa & Magambo, 397 (Haem.)
Biaggi, N., with Oliver González, Acevedo, Mariotta, Hernández Morales, Santiago Stevenson & Pérez Santiago, 54 (Hel.)
Biddle, M., with Kritzer & Kessel, 284 (Der.)
de Bie, P. & Delville, J. P., 820 (Hel.)
——, with Ghyoot & Delville, 744 (Hel.)
Biegeleisen, J. Z. & Shaffer, J. G., 734 (Am.)
——, with ——, 734 (Am.)
Bierent, P., 499 (Oph.)
Bierzynski, A., (673) (Ulc.)
Biggs, R. & Allington, M. J., 1142 (Haem.)
Biguet, J., with Coutelen & Razemon, (275) (Hel.)
Binder, T., with Sicé, 780 (Mal.)
Binet, L., Tanret, P. & Bochet, M., (877) (Bl.)
Biocca, E., (904), 1024 (Hel.)
Birnbaum, D., with Kleeberg, 265 (Am.)
——, with Kleeberg, 263 (Am.)
Bishop, A. & McConnachie, E. W., 1085 (Mal.)
Biswas, T. C., with Ghosh, 348 (Tryp.)
Bitschai, J., 568 (Hel.)
Blaché, R., with Montestruc, 270, 1127 (Lep.)
Black, R. H., 323 (Mal.)
Blair, D. M., 1073 (Mal.)
Blakely, E. R. & Reid, J. D., 661 (Hel.)
Blanc, F. & Siguier, F., 217 (B.R.)
Blanc, G. & Bruneau, J., 202 (Prot.), 1115 (R.F.)
Blanco, M. C., Resta, C. A. & Pittaluga, J. D., 364 (Am.)
Blanco Rabassa, E., with Basnuevo & Cowley Chávez, (1111) (Am.)
——, with ——, ——, Sotolongo & Achkar, 1137 (Hel.)
Blanton, F. S., Travis, B. V., Smith, N. & Husman, C. N., 91 (Ent.)
Blattner, R. J., 980 (Typh.)
Blauw, A. S., with Keegan & Anderson, 401 (Vms.)
Blitstein, I., (830) (Haem.)
Bloise, W., with Meira & Behmer, 1010 (Hel.)
de Blomenfield, E., with Vegas, Convit & Antonio Medina, 651 (Lep.)
Blümlein, H., 667 (Hel.)
Boag, T. J., with Brown, Green & Kuitunen-Ekbaum, 475 (Hel.)
Boase, A. J., 497 (Oph.)
Bocchetti, G., 1023 (Hel.)
Bochet, M., with Binet & Tanret, (877) (Bl.)
Bodenstein, O. F., with Fales, Nelson & Fulton, 1158 *bis* (Ent.)
Böe, J., Lindén, V. & Tveit, J., 754 (Der.)
Boenjamin, R., 1122 (Lep.)
Boero, H. M., 486 (Hel.)
Bogaert, W., (907) (Hel.)
Bohart, R. M., with Hsiao, 588 (Ent.)
Boiron, H., with Durieux, 156, (811) (R.F.)
Boivin, A., with Devignat, 984 (Pl.)
Bokil, K. V., with Keskar, (566) (Lep.)
Bolgert, M., Montel, L. R. & Mollinedo, R., 45 (Lep.)
Bolivia: Ministerio de Higiene y Salubridad., 215 (Reports, etc.), (1063) (Mal.)
Bolliger, A., with Backhouse, 1094 (Tryp.)
—— & Macindoe, N. M., 971 (Leish.)
Bonfiglioli, H., with Negroni & Vívoli, 1039 (Der.)
Bonilla Naar, A., with Albornoz Plata, 891 (Am.)
—— & Rodríguez Velandia, O., 1107 (Am.)

Bonnet, D. D., with Gross, 548 (Pl.)
Bonne-Wepster, J. & Swellengrebel, N. H., 128 (Mal.)
Bonnin, 554 (Am.)
Bonnin, H. & Moretti, G. F., 635 (Am.)
Bonsib, R. S., 70 (Vms.)
Bool, J., with Schüffner, 908 (Hel.)
——, with —— & Swellengrebel, 61 (Hel.)
Bordas, E. & Downs, W. G., 779 (Mal.)
——, with ——, 1082 (Mal.)
——, with —— & Navarro, 1157 (Ent.)
Borell, I. R., 1098 (Typh.)
de Borinquen Segundo, O., with Asenjo, Muñiz & Canals, (390) (Def. Dis.)
Borzone, R. A., 794 (Tryp.)
——, Lapieza Cabral, P., Alvarez de Toledo, J. M. & Aizemberg, M., 349
Bose, J. P., with Chakravarty, Sen Gupta & De, 136 (Leish.)
Bose, R., with Dharmendra & Chatterjee, 564 (Lep.)
——, with ——, Dey & Kapur, 374 (Lep.)
Botman, T., 687 (Reports, etc.)
Böttner, H., 469 (Am.)
Bouilloc, J., with Crosnier, Darbon, Ducourneau, Martin & Ricossé, 991 (Am.)
Boulard, C., with Aubry, Massonat & Zevaco, (902) (Hel.)
——, with ——, Portier, Messerschmitt & Massonnat, (919) (Haem.)
Bouquet, A. & Kerharo, J., (390) (Def. Dis.)
Bourdié, M., with Roubaud, 344 (Tryp.)
Bourgain, M., 471, 641 (R.F.)
Bouroncle, B. A., with Wright, Dodd, Doan & Zollinger, (579) (Haem.)
Boutet, R., with Galliard, 720, 968 (Tryp.)
Bovingdon, H. H. S., 763 (Ent.)
Bower, J. D., Stephens, F. F. & Wibberley, D.G., (240) (Tryp.)
Bowie, J. H., 665 (Hel).
Boxus, V., with Adant, 1045 (Misc. Dis.)
Boyd, J. S. K., (688) (Reports, etc.), 1006 (Hel.)
Boyer, J. & Corre, L., 589 (Ent.)
Bozicevich, J., with Tobie, Most & Reardon, 1110 (Am.)
Bracher, G., 250 (Typh.)
Bradin, J. L., Jr., with Faust, Sadun, Everritt & Lewis, 148 (Am.)
——, with Sadun & Faust, 1112 (Am.)
Bradley, G. H. & Lyman, F. E., 19 (Mal.)
Bradner, W. T. & Rawson, G. W., 990 (Am.)
Bradshaw, R. H., with Klein, 670 (Vms.)
Brady, F. J., with Haskins & Luttermoser, 41 (Am.)
Braga, N. de P., with Carvalho, Strauss, Arminante, Altenhein, Rocha & Vieira, 1136 (Hel.)
Branco, F., with Fonseca, Pinto, Colaço, Oliveira da Gama, Franco & Lacerda, 980 (Typh.)
von Brand, T., Baernstein, H. D. & Mehlman, B., 479 (Hel.)
——, Tobie, E. J. & Mehlman, B., 530, 964 (Tryp.)
Brandis, H. & Pöhn, H. P., 552 (Am.)
Branicki, J., (173) (Hel.)
Bras, G., with Lie Kian Joe, 56, 172 (Hel.)
Brass, W., with Foy & Kondi, 918 (Haem.)
Bray, G. T., 209 (Ent.)
Brazzaville [A. E. F.], 529 *bis* (Tryp.)
Brede, H. D., 491 (Hel.)
Brennan, J. M., 515 (Mal.)
Brent, M. M., with Balamuth, 634 (Am.)
Breslin, D., with Rigdon, 234 (Mal.)
——, with —— & Micks, 714 (Mal.)
Brisou, J., (27) (Typh.)
Britto, R. S. & Cotrim, J., 235 (Mal.)
Brizard, H., with Giroud, Le Gac & Laurent, 626, 980 (Typh.)
Brodie, B. B. & Udenfriend, S., 14 (Mal.)
Brodine, C. E., with Becker & Schwink, 1084 (Mal.)
Brody, G., with Manwell, 335 (Mal.)
de Broekert, W., 267 (Lep.)
Brooke, M. M., with Donaldson, 436 (Mal.)
Brooks, A. G., 804 (Rab.)
Brotto, W., 47 (Lep.)
Brouet, G., Marche, J. & Leprat, J., 657 (Hel.)
Brounst, G. & Anstre, B., 552 (Am.)
Brown, A. W. A., 500 (Ent.)
——, with Arnason, Fredeen, Hopewell & Rempel, (92) (Ent.)
——, with Harvey, (593) (Ent.)
——, with Orser, (594) (Ent.)
Brown, M., Green, J. E., Boag, T. J. & Kuitunen-Ekbaum, E., 475 (Hel.)
Brožek, J., with Keys, Henschel, Mickelsen, Taylor, Simonson, Skinner & Wells, 767 (B.R.)
Bruce-Chwatt, L. J., 710 (Mab.)
—— & Fitz-John, R. A., 859 (Mal.)
Brueckner, A. L., with Reagan, 457 (Rab.)
Brumpt, L. C. & Ansari, A. R., 809 (Am.)
Bruneau, J., with Blanc, 202 (Prot.), 1115 (R.F.)
Bryan, J. F., 13 (Mal.)
Bucco, G., 12 (Mal.), 151, 637 (Am.), 161 (Lep.), 357, 358 (Den.), 1099 (Typh.)
—— & Clieffi, G., 363 (Am.)
Buck, G., Courdurier, J., Dorel, R. & Quesnel, J. J., (1097) (Leish.)
Buechler-Czaczkes, E., with Guggenheim & Halevy, 896 (R.F.)
Bueding, E., 653 (Hel.)
—— & Charms, B., (476) (Hel.)
——, with Epps & Weiner, 278 (Hel.)
—— & Peters, L., 654 (Hel.)
Buenos Aires, (563) (Lep.)
Bunster, A., with Neghme, Bertín, Donckaster, Silva, Artigas, Muñoz T., Faúndes & Basso, 484 (Hel.)
Bunting, S. J. with Harris, 191 (Haem.)
Buonomini, G., Maccarrone, A. & Motta-Diana, A., 230 (Mal.)
Burch, H. B., Salcedo, J., Jr., Carrasco, E. O., Intengan, C. L. & Caldwell, A. B., 64 (Def. Dis.)
Burch, T. A., 576 (Hel.)
Bureau Permanent Inter-Africain, 338 (Tryp.)
Burgdorfer, W., 800 (Typh.)
——, with Geigy, 1114 (R.F.)
——, ——, Gsell, O. & Wiesmann, E., 542 (Typh.)
Burger, G. C. E., 766 (B.R.)
Burgess, R. W., 839 (Ent.)
——, with Hunninen & Young, 336 (Mal.)
—— & Young, M. D., 11 (Mal.)
Burke, R. C., 755 (Der.)
Burner, A. M., with Jewett & Holt, 902 (Hel.)
Burrows, R. B., 61 (Hel.)
Burrows, W., Deupree, N. G. & Moore, D. E., 258 *bis* (Chl.)
Burtner, O. W., 744 (Hel.)

Burtt, E. & Jackson, C. H. N., 530 (Tryp.)
de Bustamante, F. M., 860 (Mal.)
—— & Guedes, A. S., 516 (Mal.)
—— & Gusmão, J. B., 679 (Ent.)
——, Lacerda, N., Paracampos, H. & Santos, O. O., 319 (Mal.)
—— & Pires, W. da M., 501 (Ent.)
——, —— & Gusmão, J. B., 617 (Mal.)
Busvine, J. R., with Tew & David, 589 (Ent.)
Butori, P., with Thiodet, Fourrier & Massonnat, 468 (Am.)
Büttiker, W., 320 (Mal.)
Buttner, A., 478 (Hel.)
Butts, D. C. A., 132 (Bl.)
Butts, J. S., with Lindquist, Roth, Yates & Hoffman, 931 (Ent.)
——, with Yates, Gjullin & Lindquist, (1049) (Ent.)
Butz, W. C., with Jacobs & Felts, 585 (Misc. Dis.)
Buxton, A. P., 981 (Y.F.)
—— with Lumsden 1102 (Y.F.)
Buxton, P. A. 103 (B.R.)
Byrne A. A. 332 (Mal.)
Bystritskij I. A. 612 (Mal.)

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Cable, R. M., 303 (B.R.)
Cader, M. H. M. A., with Rajendram & Visvalingham, 17 (Mal.)
Café, B., with de Magalhães & Rocha, 728 (Typh.)
Cain, T. L., Jr., with Deonier & McDuffie, 412 (Ent.)
Cairo, 169 (Hel.)
Caldwell, A. B., with Burch, Salcedo, Carrasco & Intengan, 64 (Def. Dis.)
Calero M., C., 729, 801 (Typh.)
California Mosquito Control Association, 292 (Ent.)
California, State of: Dept. of Pub. Health, 974 (Typh.)
Calimbas, A. L., with Garcia, 404 (Misc. Dis.)
Callender, M. E., with McCowen & Lawlis, 640 (Am.)
Callot, J. & Vermeil, C., 665 (Hel.)
Calop, R., with Jausion, Benard & Aboud, (837) (Heat Str.)
Calvo Melendro, J., (822) (Hel.)
Camain, R., with Jaujou & Adam, (208) (Ent.)
——, Navarranne, P. & Ayite, E., 819 (Hel.)
——, with Sohier & Paris, (1023) (Hel.)
Camargo, L. H., (747) (Hel.)
de Camargo, T., (1143) (Haem.)
Cambiazo, M. C., with Donoso Barros, (205) (Ent.)
Cambies, J., 154 (Am.)
Cameron, H., 797 (Typh.)
Campins, H., Scharyj, M. & Cortés, R. J., 402 (Der.)
Campos R. de C., J., 266 (Lep.)
—— & Molina S., M., 371 (Lep.)
Canals, A. M., with Asenjo, de Borinquen Segundo & Muñiz, (390) (Def. Dis.)
Canet, J., 58 (Hel.), 440 (Mal.)
—— & Jahan, P., 180 (Hel.)
Cannoo, J. M., 28 (Typh.)
Capponi, M., 385 (Hel.)
Carasso, R., with Cattan, Frumusan, Cotoni & Weill-Fage, (246) (Typh.)
Cardama, J. E., with Basombrío, Gatti & Colombo, 564 (Lep.)
Cardoso, H., with Pereira & Travassos, 979 (Typh.)
Cariola, J., Prado, R., Agosín, M. & Christen, R., 451 (Tryp.)
Carle, R., (1145) (Misc. Dis.)
Carlos Arauz, J., with Ayas, (467) (Am.)
Carlos del Campo, J., (56) (Hel.)
Carlos Radice, J. & Kaplan, S., 581 (Der.)
Carpenter, C. M., 1130 (Lep.)
Carpenter, S. J., with Galindo & Trapido, 144, 802 (Y.F.)
Carpio, D., with Garduño, 483 (Hel.)
Carrasco, E. O., with Burch, Salcedo, Intengan & Caldwell, 64 (Def. Dis.)
——, with Salcedo, Bamba, Chan, Concepcion, Jose, de Leon, Oliveros, Pascual, Santiago & Valenzuela, 494 (Def. Dis.)
Carrera, G. M., 149 (Am.)
Carri, E. L. & Schisani, J. O., 737 (Am.)
Carson, R. P., with Hamrick & Cleve, 171 (Hel.)
Carvalho, A. de A., Braga, N. de P., Strauss, A., Arminante, H., Altenhein, D., Rocha, H. S. & Vieira, P. B., 1136 (Hel.)
Casas, C. B., with Hernández Morales & Garcia Sanz, 59 (Hel.)
Casile, M., 438, 862 (Mal.), 809 (Am.)
Cassamagnaghi, A., (676) (Prot.)
Castellani, A., (583) (Ulc.)
Castesana Garcia, M., 1041 (Oph.)
Castets, H. P., with Dubarry & Dussert, (396) (Sp.)
Castorina, S., 990 (Am.)
Castro Jenkins, A., with Romero & Alvarado, 1123 (Lep.)
Cates, J. E. & McIlroy, M. B., 1089 (Tryp.)
Cathie, I. A. B., 291 (Prot.)
Catros, P., with Moretti, 632 (Am.)
Cattan, R., Carasso, R., Frumusan, P., Cotoni, P. & Weill-Fage, J. C., (246) (Typh.)
Caubet, P., with Jullien-Vieroz & Montestruc, 52 (Hel.),
Causey O. R., Deane, L. M. & Deane, M. P., (838) (Ent.)
Ceccaldi, J., 529 *bis* (Tryp.)
Ceccherini, M., with Ridi, Signorini & Davoli 799 (Typh.)
Celis S., H., with Downs & Gahan, 333 (Mal.)
Cellerino, N. A., 902 (Hel.)
Cepeda, C., with Gajardo Tobar, Apablaza, Uribe, Benavides, Vargas, Rojas, Zeldis, Lucchini & De la Fuente, 381 (Hel.)
Ceylon, 598 (Reports, etc.)
Chabaud, A. G., 681 (Ent.)
——, with Gaud, 667 (Hel.)
—— & Lanz, P., 1137 (Hel.)
Chafa, with Ansari & Mofidi, 969 (Leish.)
Chagula, W. K., with Holmes, Gee, Asea, Bhima, Evelia, Kasirye, Kiboneka, Lugazia, Majale, Mughairy, Ochieng, Gesa & Magambo, 397 (Haem.)
Chaigneau, H., with Levaditi & Vaisman, 739 (R.F.)
Chakravarti, H., 149 (Am.), 453 (Leish.)
Chakravarti, R. N., Chaudhuri, R. N. & Chakravarty, N. K., 68 (Ep. Dropsy)
—— & Sen Gupta, P. C., 350 (Typh.)
Chakravarty, A., 1108 (Am.)

Chakravarty, N. K., with Chakravarti & Chaudhuri, 68 (Ep. Dropsy)
——, with Chaudhuri, 707 (Mal.)
——, Sen Gupta, P. C., Bose, J. P. & De, U. N., 136 (Leish.)
Chakravorty, A. & Basu, U. P., 12 (Mal.)
Chamberlain, R. W., 209 (Ent.)
—— & Sikes, R. K., 293 (Ent.)
Chambers, L. A. & Clawson, J. R., 246 (Typh.)
——, Cohen, S. S. & Clawson, J. R., 246 (Typh.)
——, with —— & ——, 246 (Typh.)
Chan, G. S., with Salcedo, Bamba, Carrasco, Concepcion, Jose, de Leon, Oliveros, Pascual, Santiago & Valenzuela, 494 (Def. Dis.)
Chandler, A. C., Read, C. P. & Nicholas, H. O., (383) (Hel.)
Chandra, H. & Dutt, R. L., (822) (Hel.)
Chang, A., with Chung, Chang, Feng, Lu & Tsao, 242 (Leish.)
Chang, H., with Dolman, Kerr & Shearer, 642, 994 (R.F.)
Chang, K. & Co-Workers, 275 (Hel.)
Chang, N., with Chung, 721 (Leish.)
——, with ——, Chang, Feng, Lu & Tsao, 242 (Leish.)
—— & Liu, F., 971 (Leish.)
——, Zia, S. H., Liu, F. & Chao, S., 729 (Typh.)
Chang, S. L., 469 (Am.)
Chang, Y., 894 (Am.)
Chao, S., with Chang, Zia & Liu, 729 (Typh.)
Chardome, M., with van den Berghe, 1067 (Mal.)
Charms, B., with Bueding, (476) (Hel.)
Charters, A. D., 933 (Reports, etc.)
Chartres, J. C., 418 (Reports, etc.)
Chassain, J., with Kernevez, 959 (Tryp.)
Chatterjee, K. R., with Dharmendra & Bose, 564 (Lep.)
Chatterji, S. N., with Dharmendra, 163 (Lep.)
——, with —— & Sen, 164 (Lep.)
Chaudhuri, R. N., 1070 (Mal.)
——, with Chakravarti & Chakravarty, 68 (Ep. Dropsy)
—— & Chakravarty, N. K., 707 (Mal.)
——, Ghosal, S. & Rai Chaudhuri, M. N., 255 (Chl.)
——, Rai Chaudhuri, M. N., Ghosh, S. & Dutta B. N., 711 (Mal.)
Chaussinand, R., 937 (B.R.)
—— & Besse, P., (1001) (Lep.)
Chavarría C., M., (1133) (Hel.)
Cheek, E. B., 397 (Haem.)
Chen, C., (823) (Hel.)
Chen, H. H. & Wu, P. N., 659 (Hel.)
Chen, T. H., with Quan & Meyer, 254 (Pl.)
Ch'en, W., with Liu & Koo, (1038) (Haem.)
Chenault, J. W., with Quinland, (72) (Der.)
Chenebault, J., 173 (Hel.)
Chervinskij, I. A., 610 (Mal.)
Chesterman, C. C., 81 (Misc. Dis.), 505 (B.R.), 763 (Reports, etc.), 1006 (Hel.), 1090 (Tryp.)
Chick, H., 750 (Def. Dis.)
Chida, T., with Asano, Kameda, Tamemasa & Ishii, 1086 (Mal.)
Chieffi, G., with Bucco, 363 (Am.)
——, with Gambardella, 993 (Am.)
——, with Parenzan, 1051 (Ent.)
Chipiakoff, V., 834 (Vms.)
Chiriboga, J., 194 (Der.)
——, with Croxatto, 996 (Lep.)
Chitty, D. & Kempson, D. A., 252 (Pl.)
Chiu, Y. S., with Koenigstein & Tsai, 533 (Leish.)
Chiyuto, S., (266) (Lep.)
Chodja-Zade, with Ansari & Mofidi, 351 (Leish.)
Choubrac, P., with Siguier, Piette & Herman, 155, 468 (Am.)
——, with ——, —— & Sebaoun, 808 (Am.)
Choumara, R., with Farinaud, 6 (Mal.)
——, with —— & Royer, 6 (Mal.)
Chow, C. Y., (205) (Ent.)
—— & Huang, T. C., 930 (Ent.)
——, —— & Yue, T. F., 759 (Ent.)
—— & Mattingly, P. F., (1151) (Ent.)
Choza Ferrer, J. A. & Pinero Carrillo, J .F., 106 (Mal.)
Christen, R., with Agosin & Rubio, 156, 471 (Am.)
——, with Cariola, Prado & Agosín, 451 (Tryp.)
——, with Jarpa, Agosin & Pizzi, 1096 (Tryp.)
——, with Neghme, Agosin & Rubio, 1114 (Am.)
Christen, A., R., with Agosín, Jarpa & Atías, 720 (Tryp.)
Christensen, P. A. & Widdicombe, M., 281 (Vms.)
Christiansen, M. & Siim, J. C., 1150 (Prot.)
Chu, H., with Ho & Yuan, 794 (Leish.)
Chung, H., Chang, A., Chang, N., Feng, S., Lu, J. & Tsao, Y., 242 (Leish.)
—— & Chang, N., 721 (Leish.)
Chung-Hoon, E. K., 161 (Lep.)
Ciaccio, J., with Giroud & Bablet, 1098 (Typh.)
Cicconardi, A., 233 (Mal.)
Cilesiz, A., with Berke, 457 (Rab.)
Ciuca, M., 441 (Mal.)
Claisse, R., di Matteo, J., Deuil, R. & de Traverse, P. M., 970 (Leish.)
Clark, E. W. & Morishita, F., 502 (Ent.)
Clark, J. C., with McDuffie, Eddy & Husman, 415 (Ent.)
Clark, M., 1141 (Def. Dis.)
Clark, R. W., with Smith, Saito, Beard, Kepp & Eddie, 73 (Der.)
Clark, W. H., with Lennette, 543 (Typh.)
——, Lennette, E. H. & Meiklejohn, G., 626 (Typh.)
Clarke, G. H. V., 670 (Der.)
Clarke, M. C., 599 (Reports, etc.)
Clawson, J. R., with Chambers, 246 (Typh.)
——, with —— & Cohen, 246 (Typh.)
——, with Cohen & Chambers, 246 (Typh.)
Clements, F. W. with Hipsley, 842 (B.R.)
Clemo, G. R. & Popli, S. P., (864) (Mal.)
Cleve, E. A., with Hamrick & Carson, 171 (Hel.)
Coatney, G. R., Cooper, W. C., Eyles, D. E., Culwell, W. B., White, W. C. & Lints, H. A., 124 (Mal.)
——, with ——, Jeffery & Imboden, 439 (Mal.)
——, —— & Young, M. D., 518 (Mal.)
——, with Greenberg & Trembley, 620 (Mal.)
——, with Imboden, Cooper & Jeffery, 523 (Mal.)
——, with Taylor, Greenberg & Highman, 639 (Am.)
Cochrane, R. G., 373, 565, (648) (Lep.)
Coda, D., (326), 780 *bis* (Mal.)
——, with Correa & Lima, 114 (Mal.)
Codeleoncini, E., with d'Ignazio, 139 (Typh.)
Coelho, G., 641 (Am.)
Coghill, N. F., 557 (R.F.)
Cohen, S., with Pike & Murray, 546 (Typh.)
——, with Sleisenger & Murray, 728 (Typh.)

Cohen, S. S., 246 (Typh.)
——, Chambers, L. A. & Clawson, J. R., 246 (Typh.)
——, with —— & ——, 246 (Typh.)
Coito, A. de M. F., with Nogueira, 384 (Hel.), 419 (Reports, etc.)
——, with —— & Ferreira, 280 (Haem.)
Colaço, A. F., with Fonseca, Pinto, Oliveira, Branco, da Gama, Franco & Lacerda, 980 (Typh.)
Colaço, A. F. T., with de Azevedo, 378 (Hel.)
Colbourne, M. J., 763 (Reports, etc.)
——, Edington, G. M. & Hughes, M. H., with Ward-Brew, A., 685 (Reports, etc.)
Colglazier, M. L. & Enzie, F. D., (743) (Hel.)
Colless, D. H., 94 (Lab.)
Collier, H. O. J. & Hall, I. F., 890 *bis* (Chl.)
—— & Waterhouse, P. D., 254 (Chl.)
Collins, D. L. & Nardy, R. V., 681 (Ent.)
Colombo, C. V., with Basombrío, Gatti & Cardama, 564 (Lep.)
Colón, J. E., with Asenjo & Hernández-Morales, 574 (Hel.)
Colonial Office, 409 (Ent.), 503 (Reports, etc.), 718 (Tryp.)
Colonial Products Advisory Bureau, 209 (Ent.)
Colorado Iris, R., Treviño, A., Domínguez, R. & Mazzotti, L., 55 (Hel.)
Colucci, C. F., with Jacono, 166 (Lep.)
Coluzzi, A. & Raffaele, G., 1154 (Ent.)
Comité des noms français, Entomologie, Université de Montréal, (93) (Ent.)
Conan, N. J., Jr., 555 (Am.)
—— & Hyman, G. A., 497 (Der.)
Concepcion, I., with Salcedo, Bamba, Carrasco, Chan, Jose, deLeon, Oliveros, Pascual, Santiago & Valenzuela, 494 (Def. Dis.)
——, with Uyguanco & Rodriguez, 997 (Lep.)
Condorelli, L., 1057 (B.R.)
Condorelli, V., with Previtera, (27) (Typh.)
Conejos, M., with Nájera, (745) (Hel.)
Conférence Interafricaine sur l'Alimentation et la Nutrition, 389 (Def. Dis.)
Congo Belge, 594, 595 *bis* (Reports, etc.)
Connellan J. D., with Hunter, Dillahunt, Faust, Modic, Bennett & Ingalls, 570 (Hel.)
Connolly, E. M., 1125 (Lep.)
Conrad, A. H., Jr., Manson, R. C. & Peterkin, G. A. G., 623 (Leish.)
Constant, Y., with Floch, 835 (Der.)
——, with —— & Destombes, 158, 472 (R.F.)
Convit, J., with Vegas, Antonio Medina & de Blomenfield, 651 (Dep.)
Coombs, R. R. A. & Stoker, M. G. P., 886 (Typh.)
Coonoor, 599 (Reports, etc.)
Cooper, W. C., with Coatney, Eyles, Culwell, White & Lints, 124 (Mal.)
——, Coatney, G. R., Jeffery, G. M. & Imboden, C. A., Jr., 439 (Mal.)
——, with —— & Young, 518 (Mal.)
——, with Imboden, Coatney & Jeffery, 523 (Mal.)
Cooper, Z., with Lamb, Shelmire, Morgan & Keaty, 71 (Der.)
Cope, E. J., with Kasper, Lyon & White, 361 (Am.)
Corcos, A., with Dana & Corcos, 436 (Mal.)
Corcos, V., with Dana & Corcos, 436 (Mal.)
Cordero, F. A., 623 (Leish.)
Cordero Soroa, A., 813 (Lep.)
Corkill, N. L., 68 (Vms.), 390 (Def. Dis.)
——, Creditor, H. & Stewart, G. E. S., 390 (Def. Dis.)
Cornejo, P. M., (804) (Rab.)
Corradetti, A., 619 (Mal.)
—— & Verolini, F., 437 (Mal.)
Corre, L., with Boyer, 589 (Ent.)
Corrêa, R. R., 237, 435, (435) *bis* (Mal.)
——, Lima, F. O. & Coda, D., 114 (Mal.)
Cort, W. W., 482 (Hel.)
Cortés, R. J., with Campins & Scharyj, 402 (Der.)
da Costa, A. M. & Bassères, M. S., 19 (Mal.)
Costa, B., 378 (Hel.)
de Coster, P. & Rodhain, J., 1050 (Ent.)
Cotoni, P., with Cattan, Carasso, Frumusan & Weill-Fage, (246) (Typh.)
Cotrim, J., with Britto, 235 (Mal.)
Courdurier, J., with Buck, Dorel & Quesnel, (1097) (Leish.)
Courtois, L., with Wanson & Bervoets, 414 (Ent.)
Coutelen, F., 745 (Hel.)
——, Razemon, P. & Biguet, J., (275) (Hel.)
Coutinho, J. O., 479 (Hel.)
—— & Coutinho, M. A., 569 (Hel.)
Coutinho, M.A., with Coutinho, 569 (Hel.)
Couto, C., 189 (Def. Dis.)
Covell, G., with Shortt, Fairley, Shute & Garnham 701 (Mal.)
Cowen, D. & Wolf, A., 289 (Prot.)
Cowley Chávez, O., with Basnuevo & Blanco Rabassa, (1111) (Am.)
——, with ——, Sotolongo, Blanco Rabassa & Achkar, 1137 (Hel.)
Cowper, S. G., with Azim, 568 (Hel.)
Craig, C. F., 261 (Am.)
—— & Faust, E. C., 1159 (B.R.)
Crawford, L. V., with Etteldorf, 486 (Hel.)
Creditor, H., with Corkill & Stewart, 390 (Def. Dis.)
Crewe, W. & O'Rourke, F. J., 907 (Hel.)
Crockett, C. L., Jr., with Shotton & Leavell, 735 (Haem.)
Croissant, O., with Lépine, 1104 (Rab.)
Cropper, C. F. J., 634 (Am.)
Crosnier, J., with Siguier & Piette, 38 (Am.)
Crosnier, R., Darbon, A., Ducourneau, P., Bouilloc, J., Martin, M. & Ricossé, J., 991 (Am.)
Cross, H. F., with Applewhite, 1051 (Ent.)
Cross, J. B., 837 (Prot.)
Crow, J. F., with Weiner, (763) (Ent.)
Crowell, H. H., with Morrison, Lauderdale & Mote, (590) (Ent.)
Crowell, R. L. & Fay, R. W., 682 (Ent.)
Crowther, A. F., Curd, F. H. S., Davey, D. G., Hendry, J. A., Hepworth, W. & Rose, F. L., (864) (Mal.)
——, —— & Rose, F. L., (864) (Mal.)
Croxatto, O. C. & Chiriboga, J., 996 (Lep.)
Cruickshank, E. K., 392 (Def. Dis.)
Crusz, H. & Sivalingam, V., 667 (Hel.)
Cuboni, E., 837 (Prot.)
Cuff, R. L., with Laake, Howell, Dahm & Stone, (590) (Ent.)
Cullumbine, H., 76 (Heat Str.)
Culwell, W. B., with Coatney, Cooper, Eyles, White & Lints, 124 (Mal.)

Curd, F. H. S., with Crowther, Davey, Hendry, Hepworth, & Rose, (864) (Mal.)
——, with —— & Rose, (864) (Mal.)
Cutkomp, L. K., Hess, A. D. & Keener, G. G., 297 (Ent.)

D

Dacie, J. V., 621 (Bl.)
Dagradi, A. E., Sollod, N. & Friedlander, J. H., (579) (Haem.)
Dahl, A. H., with Magy & Geib, 678 (Ent.)
Dahm, P. A., with Laake, Howell, Stone & Cuff, (590) (Ent.)
——, with Teotia, 762 (Ent.)
Dakshinamurty, S. & Sharma, M.I.D., 676 (Ent.)
Dalmat, H. T., (592) (Ent.)
Damasceno, R. G. & Arouck, R., 839 (Ent.)
——, with Deane, & Deane, (838) (Ent.)
——, with —— & Vernin, 114 (Mal.)
——, with Galvão, 19 (Mal.)
Damluji, S. F., (809) (Am.)
Dana, R., Corcos, A. & Corcos, V., 436 (Mal.)
Darbon, A., with Crosnier, Ducourneau, Bouilloc, Martin & Ricossé, 991 (Am.)
Darby, W. J., 830 (Def. Dis.)
Darrow, E. M., with Gingrich, 587 (Prot.)
Das, A. & Sen Gupta, P. C., 350 (Leish.)
Dassanayake, W. L. P., 487 (Hel.)
Dassen, R. & di Stefano, R., 371 (Lep.)
Daver, M. B., (189) (Def. Dis.)
Davey, D. G., with Crowther, Curd, Hendry, Hepworth & Rose, (864) (Mal.)
Davey, T. F., with Lowe, 1124 (Lep.)
David, A., with Jaswant Singh & Ramakrishnan, 878 (Tryp.)
David, C. V., with Raman & Ramamurthi, 55 (Hel.)
David, W. A. L., (933) (Ent.)
——, with Tew & Busvine, 589 (Ent.)
Davies, D. M., 592 (Ent.)
Davis, G. E., 593 (Ent.)
Davis, W. A., 537 (Typh.)
Davis, W. L., with Sutliff & Dulaney, 11 (Mal.)
Davoli, R., with Del Turco & Signorini, 799 (Typh.)
——, with Ridi, Signorini & Ceccherini, 799 (Typh.)
—— & Signorini, L. F., 800 *bis* (Typh.)
——, with Simondi & Signorini, 799 (Typh.)
De, U. N., with Chakravarty, Sen Gupta & Bose, 136 (Leish.)
Deane, L. M., with Causey & Deane, (838) (Ent.)
——, Deane, M. P. & Damasceno, R. G., (838) (Ent.)
——, Freire, E. P. S., Tabosa, W. & Ledo, J., 19 (Mal.)
——, Vernin, C. S. & Damasceno, R. G., 114 (Mal.)
Deane, M. P., 818 (Hel.)
——, with Causey & Deane, (838) (Ent.)
——, with Deane & Damasceno, (838) (Ent.)
Debré, R., Bertrand, I., Bargeton, E. & Mozziconacci, P., 1147 (Prot.)
Decourt, J., Giroud, P. & Goulon, M., 728 (Typh.)
Dejou, L., Jonchère, H., Koerber, R., Labail, P. & d'Almeida, J., 666 (Hel.)
Delanoë, E., 923 (Oph.)
DeLay, P. D., Lennette, E. H. & DeOme, K. B., 140 (Typh.)
Delesalle, D., with Gaud, 401 (Vms.)
Delrieu, J., with Gaud & Mechali, 433 (Mal.)
Delville, J. P., with de Bie, 820 (Hel.)
——, with Ghyoot & de Bie, 744 (Hel.)
Dennis, E. W., with Berberian & Pipkin, 154 (Am.)
DeOme, K. B., with DeLay & Lennette, 140 (Typh.)
deOng, E. R., Peer, K. C., & Fancher L. W., 297 (Ent.)
Deonier, C. C., Cain, T. L., Jr. & McDuffie, W. C., 412 (Ent.)
—— & Gilbert, I. H., 412 (Ent.)
Deriu, G., and Piastu, C. 148, 466 (Am.)
Desai, S. D., with Bhende, Purandare, Banker & Figueredo, 452 (Leish.)
Deschiens, R., 762 (Ent.)
—— & Lamy, L., 630 (Am.)
—— & Poirier, M., 571, 658 (Hel.)
Desmonts, with Lelong, Le Tan Vinh & Dupré-Bouteloup, 1147 (Prot.)
Desnues, P., with Moretti, 638 (Am.)
Destombes, P., with Floch, 267, 375, 565, (565), 645, 648, 1000 (Lep.)
——, with —— & Constant, 158, 472 (R.F.)
——, with —— & Lecuiller, 742, 999 *bis* (Lep.)
Deuil, R., with Claisse, di Matteo & de Traverse, 970 (Leish.)
Deupree, N. G., with Burrows & Moore, 258 *bis* (Chl.)
Deutschman, Z., with Freyche, 138 (Typh.)
Devignat, R., 986 (*bis*) (Pl.)
—— & Boivin, A., 984 (Pl.)
——, Legros, A. & Vercammen-Grandjean, P., 993 (Am.)
Dey, N. C., with Dharmendra, Bose & Kapur, 374 (Lep.)
Dharmendra, 163, 164 (Lep.)
——, Chatterjee, K. R. & Bose, R., 564 (Lep.)
—— & Chatterji, S. N., 163 (Lep.)
——, Dey, N. C., Bose, R. & Kapur, P. L., 374 (Lep.)
—— & Mukerjee, N., 165 (Lep.)
——, Sen, N. & Chatterji, S. N., 164 (Lep.)
Diallo, J., 718 (Tryp.)
Dias, J. A. T. S., (296) (Ent.), (449) (Tryp.)
Diaz Rivera, R. S., Pons, E. R., Jr. & Ramirez, E., 404 (Misc. Dis.)
Dick, A. M., (756) (Der.)
Dick, G. W. A., 802 (Y.F.)
——, with Gillett, Ross, Haddow & Hewitt, 627 (Y.F.)
Diercks, F. H., with Smadel, Ley & Traub, 355 (Typh.)
——, with ——, ——, ——, Tipton & Frick, 977 (Typh.)
Dierckx, F. & Helmons, J., 398 (Haem.)
Díez Melchor, F., (1132) (Hel.)
——, with Matilla, Aparicio Garrido & Prieto Lorenzo, 176 (Hel.)
Diggs, L. W., with Mickler, (580) (Haem.)
Dighiero, J., with Piaggio Blanco, Dubourdieu, Joaquin Canabal & Grosso, (56) (Hel.)
Dillahunt, J. A., with Hunter, Faust, Modic, Connellan, Bennett & Ingalls, 570 (Hel.)
Dineen, J., with Pijoan & Payne, 758 (Oph.)
Ding, P., with Minning, 748, 1035 (Hel.)
Diniz, O., 166 (Lep.)

Dixit, D. T., with Subramanian & Vaid, 126 (Mal.)
Dixon, D. S., 1044 (Ulc.)
Doan, C. A., with Wright, Dodd, Bouroncle & Zollinger, (579) (Haem.)
Doby, J. M., with Vermeil, 9 (Mal.)
Dodd, M. C., with Wright, Bouroncle, Doan & Zollinger, (579) (Haem.)
Dogra, J. R., 34 (Rab.)
Dolman, C. E., Kerr, D. E., Chang, H. & Shearer, A. R., 642, 994 (R.F.)
Domínguez, R., with Colorado Iris, Treviño & Mazzotti, 55 (Hel.)
Donaldson, A. W. & Brooke, M. M., 436 (Mal.)
Donatien, A., Gayot, G. & Poul, J., (804) (Rab.)
Donckaster, R., with Neghme, Bertín, Silva, Artigas, Bunster, Muñoz T., Faúndes & Basso, 484 (Hel.)
Donegani, J. A., Ibrahim, K. A., Ikin, E. W. & Mourant, A. E., (280) (Haem.)
Donoso, R., with Mackinnon, Artagaveytia-Allende, Vinelli, Niño, Ferrada-Urzúa & Alonso, 582 (Der.)
Donoso Barros, R. & Cambiazo M., C., (205) (Ent.)
Donoso Infante, A., Amenabar Castro, E. & Badinez, O., (809) (Am.)
——, —— & Goyeneche Petit, A., 150 (Am.)
Dorel, R., with Buck, Courdurier & Quesnel, (1097) (Leish.)
Dos Santos, M. C. F., with Muniz, 965, 966 (Tryp.)
Doull, J. A., (269), 1129 (Lep.)
Dowling, M. A. C., 327 (Mal.)
Downs, W. G., 609 (Mal.)
—— & Bordas, E., 1082 (Mal.)
——, with ——, 779 (Mal.)
——, —— & Navarro, L., 1157 (Ent.)
——, Celis S., H. & Gahan, J. B., 333 (Mal.)
Drell, W., with Schinazi, Ball & Dunn, 131 (Mal.)
Dreyfuss, F. & Benyesch, M., 831 (Haem.)
Dricot, C., 595 *bis* (Reports, etc.)
Drinnon, V. P., with Dubin & Laird, 334 (Mal.)
——, with Laird & Dubin, 335 (Mal.)
Drucker, I., 23 (Mal.)
Drukker, W. & de Vries, S. I., 119 (Mal.)
Drummond, J. C., 766 (B.R.)
——, Wilder, R. M., King, C. G. & Williams, R. R., 767 (B.R.)
Dubarry, J. J., Castets, H. P. & Dussert, A., (396) (Sp.)
Dubin, I. N., Laird, R. L. & Drinnon, V. P., 334 (Mal.)
——, with —— & ——, 335 (Mal.)
Dubois, A. & Fester, A., 270 (Lep.)
—— & Swerts, L., 741 (Lep.)
DuBois, K., (594) (Ent.)
Dubourdieu, J. J., with Piaggoi Blanco, Dighiero, Joaquin Canabal & Grosso, (56) (Hel.)
Dubreuil, with Fontan, Verger, Bentégeat & Martin, (367) (R.F.)
DuBreuil, S., with Falco & Hitchings, (949) (Mal.)
Ducourneau, P., with Crosnier, Darbon, Bouilloc, Martin & Ricossé, 991 (Am.)
Duggan, A. J. & Hutchinson, M. P., 957 (Tryp.)
Dulaney, A. D., with Sutliff & Davis, 11 (Mal.)
Dunn, M. S., with Schinazi, Drell & Ball, 131 (Mal.)
Dupré-Bouteloup, L., with Lelong, Le Tan Vinh & Desmonts, 1147 (Prot.)
Durand, P., 538, (727) (Typh.)
—— & Mathis, M., 715 (Mal.)
Duren, A. N., 1059 (Mal.)
Durieux, C., 417 (Reports, etc.)
—— & Boiron, H., 156, (811) (R.F.)
Duroux, A., Tabusse & Marty, J., (274) (Hel.)
Dussert, A., with Dubarry & Castets, (396)(Sp.)
Dutt, R. L., with Chandra, (822) (Hel.)
Dutta, A. T., Ghosh, B. K. & Gupta, J. C., 809 (Am.)
Dutta, B. N., with Chaudhuri, Rai Chaudhuri & Ghosh, 711 (Mal.)
Dy, F. J., with Smith, 18 (Mal.)
——, with —— & Del Rosario, 112 (Mal.)
Dymowska, Z., 859 (Mal.)
——, with Janicki & Konopacka, 900 (Hel.)

E

Eads, R. B., Menzies, G. C. & Ogden, L. J., (838) (Ent.)
East Africa High Commission, 935 (Reports, etc.), 951 (Tryp.), 1027 (Hel.)
Eastwood, C. G., 718 (Tryp.)
Eddie, B. U., with Smith, Saito, Beard, Kepp & Clark, 73 (Der.)
Eddy, G. W., with McDuffie, Clark & Husman, 415 (Ent.)
Edelman, M. H., with Spingarn, 260, 733 (Am.)
Edeson, J. F. B., with Field, 337 (Mal.)
Edgcomb, J. H., Arnold, J., Yount, E. H., Jr., Alving, A. S., Eichelberger, L., Jeffrey, G. M., Eyles, D. & Young, M. D., 440 (Mal.)
Edington, G. M., with Colbourne, Hughes & Ward-Brew, 685 (Reports, etc.)
Egidio, L., see Lipparoni, E.
Eichbaum, F. W., Koch-Weser, D. & Leao, A. T., 1023 (Hel.)
Eichelberger, L., with Edgcomb, Arnold, Yount, Alving, Jeffery, Eyles & Young, 440 (Mal.)
Eichholtz, F. & Hotovy, R., 187 (Hel.)
Eichler, W., 948, 1063 (Mal.)
——, Löpmann, A. & Pagast, F., 1064 (Mal.)
—— & Pagast, F., 15 (Mal.)
Eichna, L. W., Park, C. R., Nelson, N., Horvath, S. M. & Palmes, E. D., 403 (Heat Str.)
Eichold, S., with Huntington & Scott, 488 (Hel.)
Eirich, F., with Perutz & Liquori, (1142) (Haem.)
Ejercito, A., 15 (Mal.)
—— & Urbino, C. M., 703 (Mal.)
Elbel, R. E., (761) (Ent.)
Elejalde, G., with Meira & Pontes, 821 (Hel.)
Elias Latorre, A., with Suarez, Martinez & Suarez, 916 (Sp.)
Elliott, D. C., 75, 1043 (Oph.), 1122 (Lep.)
El-Sadr, A. R., 480 (Hel.)
Elsdon-Dew, R., 191 (Haem.), 463 (Am.)
—— & Freedman, L., 366 (Am.)
Elton, N. W., with Enos, 240 (Tryp.)
——, with Herrera & Nicosia, 730, 801 (Y.F.)
Enos, W. F. & Elton, N. W., 240 (Tryp.)
—— & Holmes, R. H., 927 (Misc. Dis.)
Enzie, F. D., with Colglazier, (743) (Hel.)
Epps, W., Weiner, M. & Bueding, E., 278 (Hel.)
Erfan, M. & Talaat, S., 49 (Hel.)
Erhardt, A., 899 (Hel.)
Erickson, P. T., 166, 1001, 1123 (Lep.)
——, with Johansen, 474 (Lep.)

Erkip, H., with Titiz, 30 (Typh.)
Ernst, W., 185 (Hel.)
Escobar L., P., with Velasco & Onetto, (246) (Typh.)
Espinosa, J., with Meneghello & Niemeyer, 395 (Def. Dis.)
Espinosa, J. A., (751) (Haem.)
Esther, H., (1155) (Ent.)
Etteldorf, J. N. & Crawford, L. V., 486 (Hel.)
Evans, F. C. & Freeman, R. B., 593 (Ent.)
Evans, W. J. M., 97 (Reports, etc.)
Evelia, T. R., with Holmes, Gee, Asea, Bhima, Chagula, Kasirye, Kiboneka, Lugazia, Majale, Mughairy, Ochieng, Gesa & Magambo, 397 (Haem.)
Everard, A., 37 (Am.)
Everritt, M. G., 630 (Am.)
——, with Faust, Sadun, Bradin & Lewis, 148 (Am.)
Exner, H., with Jettmar, 905 (Hel.)
Eyles, D., with Edgcomb, Arnold, Yount, Alving, Eichelberger, Jeffery & Young, 440 (Mal.)
Eyles, D. E., 237 (Mal.)
——, with Coatney, Cooper, Culwell, White & Lints, 124 (Mal.)
——, with Jeffery & Young, 704 (Mal.)
—— & Young, M. D., 231 (Mal.)

F

Fahmy, T. & Kinawy, M., (753) (Haem.)
Faiguenbaum, J., with Fanta & Neghme, (822), 822 (Hel.)
Fain, A., 174, 1021, 1029 (Hel.), 759 (Misc. Dis.)
—— & Falaise, A., (929) (Ent.)
Fairbairn, D. & Reesal, M. R., 663 (Hel.)
Fairchild, G. B. & Trapido, H., 680 (Ent.)
Fairley, N. H., 546 (Typh.), 1006 (Hel.)
——, with Shortt, Covell, Shute & Garnham, 701 (Mal.)
Fakaçelli, N. M. & Konstantinidu, S., 927 (Misc. Dis.)
Falaise, A., with Fain, (929) (Ent.)
Falco, E. A., DuBreuil, S. & Hitchings, G. H., (949) (Mal.)
——, Goodwin, L. G., Hitchings, G. H., Rollo, I. M. & Russell, P. B., 872 (Mal.)
——, Russell, P. B. & Hitchings, G. H. (949) (Mal.)
Falcone, F., with Villani, (628) (Rab.)
Falcone, G., 1040 (Oph.)
Fales, J. H., Nelson, R. H., Fulton, R. A. & Bodenstein, O. F., 1158 *bis* (Ent.)
Fancher, L. W., with deOng & Peer, 297 (Ent.)
Fanta, E., Faiguenbaum, J. & Neghme, A., (822), 822 (Hel.)
Farina, R., 371 (Lep.)
Farinaud, E., Lévy, G. & Gros, R., 674 (Misc. Dis.)
Farinaud, M. E. & Choumara, R., 6 (Mal.)
——, Lévy, G. & Frézières, H., 247 (Typh.)
——, Royer, J. & Choumara, R., 6 (Mal.)
——, with Soulage, Tauzin & Lefebvre, 253 (Pl.)
Farquhar, H. G., 407 (Prot.)
Faúndes, A., with Neghme, Bertín, Donckaster, Silva, Artigas, Bunster, Muñoz, T. & Basso 484 (Hel.)
Faure, J., 485 (Hel.)
——, with Gaud & Maurice 609 (Mal.)
Faure, R. M., 731 (Den.)
Faust, E. C. (152) (Am.) 504 (Reports etc.) 840 (B.R.) 1145 (Misc. Dis.)
——, with Craig, 1159 (B.R.)
——, with Hunter, Dillahunt, Modic, Connellan, Bennett & Ingalls 570 (Hel.)
——, with Sadun & Bradin 1112 (Am.)
——, Sadun, E. H., Everritt, M. G., Bradin, J. L. Jr. & Lewis, R. A, 148 (Am.)
——, Scott, J. A., & Taylor, J. E., 108 (Mal.)
Fawdry, A. L. & Mazhar, M., 1096 (Leish.)
Fay, R. W., with Crowell, 682 (Ent.)
——, with Jensen, 1153 (Ent.)
——, with —— & Sumerford, 416 (Ent.)
Fehler, B. M., with Saner, 356 (Typh.)
Feldman, H. A., with Sabin, 202 (Prot.)
Feldman, W. H., Karlson, A. G. & Grindlay, J. H., 1120 (Lep.)
Felix, A., 26, 546 (Typh.)
Félix Contreras, D., 817 (Lep.)
Felsenfeld, O., with Gradwohl & Benitez Soto, 1054 (B.R.)
——, Soman, D. W., Ishihara, S. J., Waters, T. & Norsen, J., 988 (Chl.)
——, ——, Young, V. M., Yoshimura, T., Waters, T. & Ishihara, S. J., 987 (Chl.)
——, Young, V. M. & Ishihara, S. J., 258 (Chl.)
Felts, J. H., with Jacobs & Butz, 585 (Misc. Dis.)
Fendall, N. R. E., 241 (Leish.)
Feng, S., with Chung, Chang, Chang, Lu & Tsao, 242 (Leish.)
Ferguson, P., with Starr & Salmon, 211 (Ent.)
Féron, J., 269, 649 (Lep.)
Ferrada Urzúa, L. V., with Mackinnon, Artagaveytia-Allende, Vinelli, Niño, Alonso & Donoso, 582 (Der.)
——, with Niño & Risolía, 74 (Der.)
Ferreira, A. E., with Nogueira & Coito, 280 (Haem.)
Ferreira, D. L., 45 (Lep.)
Ferreira, F. S. da C., 340 (Tryp.)
—— & de Almeida, C. L., (448) (Tryp.)
——, —— & Pinto, A. R., 341 (Tryp.)
——, —— & Pires, F. M., 341 (Tryp.)
—— & Tendeiro, J., 241 (Tryp.)
Ferreira, J. M. & Meira, J. A., 406 (Misc. Dis.)
Ferreira, M. O., with Rachou & Lima, 618 (Mal.)
Ferri, R. G., with Guidolin, 495 (Vms.)
Ferro-Luzzi, G., 198 (Misc. Dis.), 393, 1036 (Def. Dis.)
Fester, A., with Dubois, 270 (Lep.)
Field, J. W. & Edeson, J. F. B., 337 (Mal.)
Fiennes, R. N. T.-W., 343, 963 (Tryp.)
Figueredo, N., with Bhende, Purandare, Banker & Desai, 452 (Leish.)
Filer, M. K., with Baranger, 716 (Mal.)
Findlay, G. H., 1046 (Misc. Dis.)
Findlay, G. M., 85 (Misc. Dis.), 420 (B.R.)
——, with Ampofo, 368 *ter* (Ys.)
Finke, L., with Westphal, 200 (Prot.)
Finland, M., with Kass & Neva, 725 (Typh.)
Fischer, W. O., 199 (Misc. Dis.)
Fischl, F. M. & Sulman, F. G., 34 (Rab.)
Fisher, H. J. W., with Wyllie, 291 (Prot.)
Fite, G. L., (996) (Lep.)
Fitz-John, R. A., with Bruce-Chwatt, 859 (Mal.)
Fleckenstein, A. & Gayer, J., (281) (Vms.)
Fletcher, E., 519 (Mal.)

Floch, H., 617 (Mal.)
—— & Abonnenc, E., 348 (Tryp.), (414), (415), (680) (Ent.)
——, Barrat, R. & Abonnenc, E., 1143 (Vms.)
—— & Constant, Y., 835 (Der.)
——, —— & Destombes, P., 158, 472 (R.F.)
—— & Destombes, P., 267, 375, 565, (565), 645, 648, 1000 (Lep.)
——, —— & Lecuiller, A., 742, 999 *bis* (Lep.)
—— & Rist, N., 375 (Lep.)
Floris, M., with Lippi, 399 (Haem.)
Fluno, J. A., with Gouck, 416 (Ent.)
Fondacaro, A., with Reitano, 573 (Hel.)
Fonseca, F., Pinto, M. R., Colaço, A. F., Oliveira, J., Branco, F., da Gama, M. M., Franco, J. S. & Lacerda, M. T., 980 (Typh.)
Fontan, Verger, Bentégeat, Martin & Dubreuil, (367) (R.F.)
Forattini, O. P., with da Silva, 793 (Tryp.)
Ford, E., 4 (Mal.)
Forster, E. F. B., 134 (Tryp.)
Fort, M., with Schwetz & Baumann, (745), (1014) (Hel.), 855 (Mal.)
Fourrier, A., with Thiodet, Butori & Massonnat, 468 (Am.)
——, with ——, Pietri, Massonnat & Aubry, 992 (Am.)
Fowler, E. M., with Reese & Morrison, 142 (Bart.)
Fowler, J. C., with Ruchman, 1150 (Prot.)
Fowler, W. M., with Hamilton, (919) (Haem.)
Fox, I., 247, 625 (Typh.)
Foy, H. & Kondi, A., 528 (Bl.)
——, —— & Alexandrides, C., 918 (Haem.)
——, —— & Brass, W., 918 (Haem.)
——, ——, Hargreaves, A. & Lowry, J., 67 (Haem.)
de Francesco, F. & d'Ignazio, C., 949 (Mal.)
Francis, J. & Spinks, A., 268 (Lep.)
Franco, J. S., with Fonseca, Pinto, Colaço, Oliveira, Branco, da Gama & Lacerda, 980 (Typh.)
Frank, H. R., (353), 537 (Typh.)
Franklin, J., Wasserman, E. & Fuller, H. S., 625 (Typh.)
Franza, R., 247 (Typh.)
Fredeen, F. J. H., with Arnason, Brown, Hopewell & Rempel, (92) (Ent.)
Freedman, L., with Elsdon-Dew, 366 (Am.)
Freeman, R. B., with Evans, 593 (Ent.)
Freire, E. P. S., with Deane, Tabosa & Ledo, 19 (Mal.)
Freire, S. A., with de Magalhães & Rocha, (541) (Typh.)
de Freitas, J. L. P., 347, 450 (Tryp.)
Freyche, M. J., 757 (Oph.)
—— & Deutschman, Z., 138 (Typh.)
—— with Sidky, 75 (Oph.)
Frézières, H., with Farinaud & Lévy, 247 (Typh.)
Frick, L. P., with Smadel, Ley, Diercks, Traub & Tipton, 977 (Typh.)
Friederich, H. C., with Zenner & Beutnagel, (671) (Der.)
Friedheim, E. A. H., with Kivits, 896 (Ys.)
Friedlander, J. H., with Dagradi & Sollod, (579) (Haem.)
Frumusan, P., with Cattan, Carasso, Cotoni & Weill-Fage, (246) (Typh.)
Fuchs, A., 757 (Oph.)
De la Fuente, J., with Gajardo Tobar, Apablaza, Uribe, Benavides, Vargas, Cepeda, Rojas, Zeldis & Lucchini, 381 (Hel.)
Fuhrmann, G., 95 (Lab.)
—— & Harten, R., 1066 (Mal.)
—— & Knüttgen, H., 117 (Mal.)
Fuller, F., with Sadun, Ané & Lewis, 261 (Am.)
Fuller, H. S., with Franklin & Wasserman, 625 (Typh.)
Fulton, J. D., Joyner, L. P., King, H., Osbond, J. M. & Wright, J., 365 (Am.)
——, —— & Price, I. N. O., 734 (Am.)
Fulton, R. A., with Fales, Nelson & Bodenstein, 1158 *bis* (Ent.)
Furtado, T. A., 402 (Der.)
Fus, with Piganiol & Rajaobelina, 926 (Ulc.)

G

Gaase, A., with Trub, 913 (Hel.)
Gaberlini, J., Jr., with Rachou, 434 (Mal.)
Le Gac, P., 680 (Ent.), (883), 977 (Typh.)
——, with Giroud, Brizard & Laurent, 626, 980 (Typh.)
——, Lagarde, J. & Mulet, M., 496 (Der.)
——, with Montel & Mulet, 472, 739 (Ys.)
—— & Rouby, M., 540 (Typh.)
Gaehlinger, 894 (Am.)
Gahan, J. B., with Downs & Celis S., 333 (Mal.)
Gaines, J. C., with Robertson, (932) (Ent.)
Gajardo Tobar, R., (383) (Hel.)
——, Apablaza, H., Uribe, P., Benavides, I., Vargas, A., Cepeda, C., Rojas, E., Zeldis, A., Lucchini, A. & de la Fuente, J., 381 (Hel.)
Galindo, P., Carpenter, S. J. & Trapido, H., 802 (Y.F.)
——, Trapido, H. & Carpenter, S. J., 144 (Y.F.)
Gall, D. & Andrews, W. H. H., (594)(Lab.)
—— & Maegraith, B. G., (601) (Reports, etc.)
Galliard, H., 1024 (Hel.)
—— & Boutet, R., 720, 968 (Tryp.)
—— & Lapierre, J., 129, 869 (Mal.)
Gallut, J., 255 (Chl.)
Galvão, A. L. A. & Damasceno, R. G., 19 (Mal.)
——, with Meira, (384) (Hel.)
Galvão, P. E., 76 (Heat Str.)
da Gama, M. M., with Fonseca, Pinto, Colaço, Oliveira, Branco, Franco & Lacerda, 980 (Typh.)
Gambardella, A. & Chieffi, G., 993 (Am.)
Gambini, G., with Benetazzo, 277 (Hel.)
Gan, K. H., with Gispen & Westermann, 795 (Typh.)
Garber, E. D., with Levine, 145 (Pl.)
——, Wolochow, H. & Smith, P., 805 (Pl.)
Garcia, E. G., 1018 (Hel.)
——, with Pesigan, Basaca-Sevilla, Pangilinan, Saniel, Banzon, Beltran, Putong & Millar, 1017 (Hel.)
——, with ——, Pangilinan, Saniel, Banzon & Putong, 1018 (Hel.)
——, with ——, Putong & Millar, 1015 (Hel.)
Garcia, E. Y., 9 (Mal.)
—— & Calimbas, A. L., 404 (Misc. Dis.)
García Barón, A., (54), (58) (Hel.)
Garcia Lopez, G., with Spies, Milanes, Lopez Toca & Reboredo, 400 (Haem.)
——, with ——, ——, ——, —— & Stone, 399 (Haem.)

Garcia Sanz, M., with Hernández Morales & Casas, 59 (Hel.)
Gard, S., 675 (Prot.)
Garduño, D. M. & Carpio, D., 483 (Hel.)
—— & de Leon, D. M., 1128 (Lep.)
Garipuy, A. & Magdelenat, P., (990) (Am.)
Garnham, P. C. C., 292 (Prot.), 608 (Mal.)
——, with Shortt, Fairley, Covell & Shute, 701 (Mal.)
Gärtner, H. & Müting, L., 486, 904 (Hel.)
Gartrell, F. E. & Johnson, A. H., 129 (Mal.)
Gatti, J. C., with Basombrío & Cardama Colombo, 564 (Lep.)
Gaud, J. & Chabaud, A. G., 667 (Hel.)
—— & Delesalle, D., 401 (Vms.)
——, Faure J. & Maurice, A., 609 (Mal.)
——, with Joyeux & Baer, 173 (Hel.)
—— & Laurent, J., 512 (Mal.)
—— & Mechali, D., 713 (Mal.)
——, —— & Delrieu, J., 433 (Mal.)
——, with Pagès, (1137) (Hel.)
Gaugusch, Z., 485, 1022 (Hel.)
Gayer, J., with Fleckenstein, (281) (Vms.)
Gayot, G., with Donatien & Poul, (804) (Rab.)
Gazette Médicale de France, 461 (Am.)
Gee F. L., with Holmes, Asea, Bhima, Chagula, Evelia, Kasirye, Kiboneka, Lugazia, Majale, Mughairy, Ochieng, Gesa & Magombo, 397 (Haem.)
Gehr, E., (43) (Lep.)
Geib, A. F., with Magy & Dahl, 678 (Ent.)
Geigy, R. & Burgdorfer, W., 1114 (R.F.)
——, with ——, Gsell & Wiesmann 542 (Typh.)
Gelfand, M., 81, 83 (Misc. Dis.)
Gennis, J., with Straus, 614 (Mal.)
Gerking, S. D., with Robinson, Turrell & Kincaid, 78 (Heat Str.)
Germain, A. & Marty, J., 969 (Leish.)
Gersdorff, W. A. & Mitlin, N., (932), 1155 (Ent.)
Gesa, J. I. H., with Holmes, Gee, Asea, Bhima, Chagula, Evelia, Kasirye, Kiboneka, Lugazia, Majale, Mughiary, Ochieng & Magambo, 397 (Haem.)
Geukens, 825 (Hel.)
Geukens, A., 522 (Mal.)
Ghosal, S., with Chaudhuri & Rai Chaudhuri, 255 (Chl.)
Ghosh, B. K., with Dutta & Gupta, 809 (Am.)
Ghosh, P. K., 461 (Pl.)
Ghosh, S., with Chaudhuri, Rai Chaudhuri & Dutta, 711 (Mal.)
Ghosh, S. M., 840 (Ent.)
—— & Biswas, T. C., 348 (Tryp.)
—— & Mukherji, A., 152 (Am.)
Ghyoot, E., Delville, J. P. & de Bie, P., 744 (Hel.)
Giaquinto-Mira, M., 677 (Ent.)
Giarman, N. J., 1091 (Tryp.)
Gibson, C. D., with Rose & Kneeland, 547 (Typh.)
Gibson, R. D., with Wenzel, 664 (Hel.)
Gilani, A., with Miller, 1109 (Am.)
Gilbert, I. H., with Deonier, 412 (Ent.)
Gilbrin, E., with Hillemand & Toulet, (275) (Hel.)
Gil Collado, J., with de Prada & Mingo Alsina, 1101 (Typh.)
Gilges, W., 1115 (R.F.), 1131 (Hel.)
Gillet, J., 48 (Hel.), 564 (Lep.)
Gillett, J. D., with Ross, 627 (Y.F.)
——, Ross, R. W., Dick, G.W.A., Haddow, A. J. & Hewitt, L. E., 627 (Y.F.)
Gingrich, W. D. & Darrow, E. M., 587 (Prot.)
Girard, G., 983 (Pl.)
Giroud, P., 244 (Typh.)
——, Ciaccio, J. & Bablet, J., 1098 (Typh.)
——, with Decourt & Goulon, 728 (Typh.)
—— & Jadin, J., 544, 545, 727 (Typh.)
——, with ——, 543, 885 (Typh.)
——, —— & Jezierski, A., 1101 (Typh.)
——, le Gac, P., Brizard, H. & Laurent, G., 626, 980 (Typh.)
Gispen, R., 140, 539 (Typh.)
——, Gan, K. H. & Westermann, C. D., 795 (Typh.)
—— & Warsa, R., 975 (Typh.)
Gjullin, C. M., (90) (Ent.)
——, with Yates, Lindquist & Butts, (1049) (Ent.)
Glajchen, D., with Zumpt, 405 (Misc. Dis.)
Glover, P. E., 450 (Tryp.)
Gluckman, H., 845 (B.R.)
Goarnisson, J., 844 (B.R.)
Goble, F. C., 1095 (Tryp.)
Goldman, L. & O'Hara, H., 282 (Der.)
Golem, S.B., 884 (Typh.)
Gomes, J. M. & de Souza, L. A., 370 (Lep.)
Gomez, J. A., with van Goor & Lodens, 325 (Mal.)
Gomez Camba, J., with Matilla, Aparicio Garrido & Prieto Lorenzo, 822 (Hel.)
Gomez, Lince, L. F., with Rodriguez, M. & Montalvan, C., 382 (Hel.)
Gomez Orbaneja, J. & Puchol, J. R., 998 (Lep.)
González Castro, J., 901, 1136 (Hel.)
——, with Morell Cuéllar, 1133 (Hel.)
Gonzales Prendes, M. A. & Ybarra Perez, R., 370, 814 (Lep.)
——, with Ybarra Perez, 814 (Lep.)
Gonzalo Maximo, Q., with Hoekenga, 40 (Am.)
Goodwin, L. G., with Berks, 556 (R.F.)
——, with Falco, Hitchings, Rollo & Russell, 872 (Mal.)
——, & Walls, L. P., 345 (Tryp.)
Goodwin, M. H., Jr., 319, 716 (Mal.)
van Goor, W. T., Lodens, J. G. & Gomez, J. A., 325 (Mal.)
Gopalan, C., 585 (Misc. Dis.)
Gordon, R. M., 88 (Ent.), 177, 1006 (Hel.)
Gören, S., 69 (Vms.)
Gouck, H. K. & Fluno, J. A., 416 (Ent.)
Goulon, M., with Decourt & Giroud, 728 (Typh.)
de Gouveia, E., with Trincão, 239, 792 (Tryp.)
——, with ——, Pinto & de Almeida, 281 (Haem.)
Goyeneche Petit, A., with Donoso Infante & Amenabar Castro, 150 (Am.)
Gradwohl, R. B. H., Benitez Soto, L. & Felsenfeld, O., 1054 (B.R.)
Graf, H., with Zumpt, 295 (Ent.)
Grainger, W. E., with Heisch, 42 (R.F.)
Grall, A., 668 (Def. Dis.)
Gramberg, K. P. C. A., (566), 817 (Lep.)
Gramiccia, G., with Quraishi & Ahmed, 1064 (Mal.)
Grant, J. S., 237 (Mal.)
Graves, L. M., with Tierkel, Tuggle & Wadley, 35 (Rab.)
Gray, H. F., Peters, R. F. & Washburn, G. E., 292 (Ent.)
Green, J. E., with Brown, Boag & Kuitunen-Ekbaum, 475 (Hel.)

Greenberg, J., 838 (Ent.)
——, with Josephson, Taylor & Ray, 862 (Mal.)
—— & Richeson, E. M., 950 (Mal.)
——, with Taylor, Highman & Coatney, 639 (Am.)
——, —— & Josephson, E. S., 873 (Mal.)
——, with ——, —— & Ray, 873 (Mal.)
——, Trembley, H. L. & Coatney, G. R., 620 (Mal.)
Gregoire, C., with Schoenaers, (765) (Reports, etc.)
Gregson, J. D., 993 (R.F.)
——, with Jellison, 86 (Misc. Dis.)
Grenier, P. & Ovazza, M., 761 (Ent.)
Grennaus, G. I., Troitskij, S. A. & Miloradovskaja, B. A., 611 (Mal.)
Griffin, A. M., with McCarten, (365) (Am.)
Griffiths, R. B., 660 (Hel.)
Grimes, C., 269 (Lep.)
Grindlay, J. H., with Feldman & Karlson, 1120 (Lep.)
Grjebine, A., 928 (Ent.)
Grob, D., (594) (Ent.)
Grocott, R. G., 88 (Prot.)
Groen, J., with Reisel, 825 (Hel.)
Groman, N. B., 874 (Mal.)
Gros, R., with Farinaud & Lévy, 674 (Misc. Dis.)
Gross, B. & Bonnet, D. D., 548 (Pl.)
Grosso, O. F., with Piaggio Blanco, Dubourdieu, Dighiero & Joaquin Canabal, (56) (Hel.)
Grün, H., 156 (R.F.)
Grunberg, E. & Schnitzer, R. J., 1001 (Lep.)
Gsell, O., with Burgdorfer, Geigy & Wiesmann, 542 (Typh.)
Guedes, A. S., with de Bustamante, 516 (Mal.)
——, with Rachou & Ricciardi, 324 (Mal.)
Guerra, P., 195 (Oph.)
Guerret, M. & Legeay, G., (145) (Rab.)
Guggenheim, K., Buechler-Czaczkes, E. & Halvey, S., 896 (R.F.)
Guidolin, R. & Ferri, R. G., 495 (Vms.)
Guild, L. P., with Munsell, Williams, Troescher, Nightingale & Harris, (751) (Def. Dis.)
Guilleman, 893 (Am.)
Guillot, C. F., 72 (Der.)
Guimarães, F. N., 560 (Ys.)
——, with Lins, Vasconcellos & Miranda, 160 (Ys.)
—— & Macedo, D. G., 283 (Der.)
—— & Travassos, J., 369 (Ys.)
Guimarães, N., 497 (Der.)
Guirguis, S., with Sorour & Halim, (745) (Hel.)
Gunther, C. E. M., 523 (Mal.), 876 (Bl.)
Günther, P. G., 396 (Sp.)
Gupta, J. C., with Dutta & Ghosh, 809 (Am.)
——, with Panja & Banerji, 581 (Der.)
Gurdjian, E. S., with Latimer & Webster, 628 (Rab.)
Gurkirpal Singh & Ahuja, M. L. (359) (Chl.)
Gurri, J., with Mackinnon, 195 (Der.)
Gusmão, J. B., with de Bustamante, 679 (Ent.)
——, with —— & Pires, 617 (Mal.)
Gutch, C. F., 626 (Am.)
Gutiérrez Estarlí E. with Basnuevo 468 (809) (Am.)
Guyane Française & Inini, 563 (Lep.)
Guzman, M., with Scrimshaw & Mendez de la Vega, 827 (Def. Dis.)

H

Haas, V. H., with Hahn & Wilcox, 132 (Mal.)
Haas, W., 854 (Mal.)
Habbu, M. K., with Sokhey, 256 *ter* (Chl.)
——, with —— & Bharucha, 256 (Chl.)
Hackett, C. J., 81 (Misc. Dis.)
Hadaway, A. B. & Barlow, F., 681, 1053 (Ent.)
Haddad, E., with Scaillet, 961 (Tryp.)
Haddow, A. J., 455 (Y.F.)
——, with Gillett, Ross, Dick & Hewitt, 627 (Y.F.)
—— & van Someren, E. C. C., (293) (Ent.)
Hadler, W. A., with Mauri, (566) (Lep.)
Hafez, M., 294 (Ent.)
Hageman, R. H., with Pagan, 206 (Ent.)
Hahn, P. F., Haas, V. H. & Wilcox, A., 132 (Mal.)
—— & Offutt, E. P., 175 (Hel.)
Haines, T. W., with Lindsay, 1050 (Ent.)
Halawani, A., Abdallah, A. & El-Kordy, M. I., 155, 806 (Am.)
—— & Baz, I. I., 13 (Mal.)
——, Latif, N. & Taha, A., with Khalaf, G., 1013 (Hel.)
——, with Newsome, 48 (Hel.)
Halevy, S., with Guggenheim & Buechler-Czaczkes, 896 (R.F.)
Halim, S. A., with Sorour & Guirguis, (745) (Hel.)
Hall, E. R. & Villa, R. B., (212) (Reports, etc.)
Hall, H. E., with Bauer, Parker, Benson, Joslin, Hightower, Snyder, Venable & Woodward, 979 (Typh.)
——, with Parker, Bauer, Lister & Woodward, 540 (Typh.)
Hall, I. F., with Collier, 890 *bis* (Chl.)
Hall, W. H., 806 (Am.)
Hall, W. J., 409 (Ent.)
von Haller, E., 1057 (B.R.)
Haller, H. L., with Rohwer, (594) (Ent.)
Hamblin, D. O., with Abrams & Marchand, (594) (Ent.)
Hamed, A., 552 (Am.)
Hamilton, C. S. P., 711 (Mal.)
Hamilton, H. E. & Fowler, W. M., (919) (Haem.)
Hamrick, L. W., Jr., Cleve, E. A. & Carson, R. P., 171 (Hel.)
Hanks, J. H., 996 (Lep.)
—— & Backerman, T., 270 (Lep.)
Hannak, J., with Schmidt & Mendheim, 577 (Hel.)
Hanno, H. A. & Margolies, M. P., 398 (Haem.)
Hansen, E. L., with Anderson, 467 (Am.)
Hansens, E. J., (590) (Ent.)
Harant, H. & Huttel, W., 70 (Vms.)
Harding, J. P. & Washtell, C. C. H., 94 (Lab.)
Hardy, F., with de Meillon, 502 (Ent.)
Harel, S. B., with Trager & Stauber, 790 (Mal.)
Hargreaves, A., with Foy, Kondi & Lowry, 67 (Haem.)
Harper, J., 81 (Misc. Dis.)
Harrell, G. T., with Aikawa & Miller, 915 (Hel.)
Harrington, R. F., Spindler, L. A. & Hill, C. H. 280 (Hel.)
Harris, J. W., with Bunting, S. J., 191 (Haem.)
Harris, R. S., with Munsell, Williams, Guild Troescher & Nightingale, (751) (Def. Dis.)
Harrison, A., with Winteringham & Loveday, 501 (Ent.)
Harrison, C. M., 930 (Ent.)

Harrison, J. L., with Audy, 546 (Typh.)
Harrison, R. A., (414) (Ent.)
Hart, E. W., Paulley, J. W., Rivers, J. S. & Westlake, E. K., 1145 (Prot.)
Harten, R., with Fuhrmann, 1066 (Mal.)
Hartz, P. H., 38 (Am.), 387 (Hel.)
Hartzell, A., with Lehman & Ward, (594) (Ent.)
Harvey, G. T. & Brown, A. W. A., (593) (Ent.)
Harvey, M. S., 542 (Typh.)
Hasegawa, K., with Sasa, Kano, Hayashi, Kimura, Miura, Oyama, Sato & Hosoya, 588 (Ent.)
Hasegawa, S., with Hashimoto, Adachi & Sato, 566 (Lep.)
Hashimoto, T., Adachi, S., Sato, M. & Hasegawa, S., 566 (Lep.)
Haskins, W. T., 1132 (Hel.)
——, Luttermoser, G. W. & Brady, F. J., 41 (Am.)
Hassett, C. C., with Jenkins, (928) (Ent.)
Hatt, R. T. & Villa R., B., (212) (Reports, etc.)
Havlik, O., 730 (Bart.)
Hawking, F., 177 (Hel.)
—— & Thurston, J. P., 868 (Mal.), 1088 (Tryp.)
Hawkins, W. B., with Ludvik & Snow, 616 (Mal.)
Haworth, R. D., McKenna, J., Powell, R. G. & Woodward, P., (894) (Am.)
Hayashi, H., 883 (Typh.)
Hayashi, S., with Sasa & Kano, (293) (Ent.)
——, with ——, ——, Kimura, Miura, Oyama, Sato, Hosoya & Hasegawa, 588 (Ent.)
Hediger, H., 1161 (B.R.)
Heilmeyer, L., Müller, W. & Schubothe, H., 831 (Haem.)
Heinkel, K., 705 (Mal.)
Heisch, R. B., 98 (Reports, etc.), 157 (R.F.)
—— & Grainger, W. E., 42 (R.F.)
van Hell, J. C., 322, 514 (Mal.)
Hellerman, L., with Rees, Reardon & Bartgis, 259 (Am.)
Hellingman, J. E., with Ketelaar, (763) (Ent.)
Helmons, J., with Dierckx, 398 (Haem.)
Hempel, G., 267 (Lep.)
Henderson, A. B., 832 (Haem.)
Henderson, E., with Seneca, 359 (Ch.), 1092 (Tryp.)
Henderson, J. M., with Rehn & Maldonado Capriles, 113 (Mal.)
——, with —— & Serrano, 113 (Mal.)
Hendry, J. A., with Crowther, Curd, Davey, Hepworth & Rose, (864) (Mal.)
Hengel, R., Kausche, G. A. & Sheris, E., 249 (Typh.)
Hennig, W., 306 (B.R.)
Henschel, A., with Keys, Brožek, Mickelsen Taylor, Simonson, Skinner & Wells, 767 (B.R.)
Hentel, W., Newton, J. K. & Horsman, R. K., 671 (Der.)
Hepding, L., with Zima, v. Werder, van Schoor & Hoffman, 186 (Hel.)
Hepworth, W., with Crowther, Curd, Davey, Hendry & Rose, (864) (Mal.)
Heriberto Torres, J. & Moscoso Carrasco, C., (705) (Mal.)
Herman, C. M., 875 (Mal.)
Herman, J. M., with Siguier, Choubrac & Piette, 468 (Am.)
——, with ——, Piette & Choubrac, 155 (Am.)
Hernández Morales, F., with Asenjo & Colón, 574 (Hel.)
——, Casas, C. B. & Garcia Sanz, M., 59 (Hel.)
——, with Oliver González, Biaggi, Acevedo, Mariotta, Santiago Stevenson & Pérez Santiago, 54 (Hel.)
Herrera, J. M., Elton, N. W. & Nicosia, J., 730, 801 (Y.F.)
Herriott, R. M., with Tenbroeck, 458 (Rab.)
Hertig, M., 25 (Leish.)
Herzberg, K., 541, 885 (Typh.)
——, Herzberg-Kremmer, H. & Urbach, H., 544 (Typh.)
—— & Urbach, H., 885 (Typh.)
Herzberg-Kremmer, H., with Herzberg & Urbach, 544 (Typh.)
Hess, A. D., with Cutkomp & Keener, 297 (Ent.)
Hewitt, L. E., with Gillett, Ross, Dick & Haddow, 627 (Y.F.)
Hewitt, R., with Kenney, 488 (Hel.)
Hewlett, P. S. & Plackett, R. L., (212) (Ent.)
Heydon, G. A. M., with Backhouse, 386 (Hel.)
Hide, C. G., with Keller, 824 (Hel.)
Highman, B., with Taylor, Greenberg & Coatney, 639 (Am.)
Hightower, J. A., with Bauer, Parker, Hall, Benson, Joslin, Snyder, Venable & Woodward, 979 (Typh.)
Hill, C. H., with Harrington & Spindler, 280 (Hel.)
Hill, E. L., Morlan, H. B., Utterback, B. C. & Schubert, J. H., 976 (Typh.)
Hill, J., 130 (Mal.)
Hill, K. R., Kodijat, R. & Sardadi, M., 1117 (Ys.)
Hill, T. S., with Overman & Wong, 118 (Mal.)
Hillemand, P., Gilbrin, E. & Toulet, J., (275) (Hel.)
—— & Rosenstiel, 153 (Am.)
Himpe, N. E. & Pierquin, L., 125 (Mal.), 1116 (R.F.)
Himsworth, H. P., 503 (Reports, etc.)
Hipsley, E. H. & Clements, F. W., 842 (B.R.)
Hirte, W., 1134 (Hel.)
Hitchcock, D. J., 900 (Hel.)
Hitchcock, J. D., with Kuitert, 513 (Mal.)
Hitchings, G. H., with Falco & DuBreuil, (949) (Mal.)
——, with ——, Goodwin, Rollo & Russell, 872 (Mal.)
——, with —— & Russell, (949) (Mal.)
——, with Russell, (949) (Mal.)
Ho, E. A., Yuan, I. & Chu, H., 794 (Leish.)
Hoare, C. A., 241 (Tryp.)
Hocking, B., 592 (Ent.)
——, Richards, W. R. & Twinn, C. R., (90) (Ent.)
——, Twinn, C. R. & McDuffie, W. C., 208 (Ent.)
Hodgkin, E. P., 10 (Mal.)
——, with Reid, 321 (Mal.)
Hoekenga, M. T., 48, 1134 (Hel.), 863 (Mal.), 1109 (Am.)
—— & Gonzalo Maximo, Q., 40 (Am.)
—— & Tucker, H. A., 285 (Der.)
Hoeppli, R., (1002) (Hel.)
Hoffman, R. A., with Lindquist, Roth, Yates & Butts, 931 (Ent.)
Hoffmann, A., with Zima, v. Werder, van Schoor & Hepding, 186 (Hel.)
Hogan, T. W., (1158) (Ent.)
Hogg, E. S., 58 (Hel.)

Holden, W. S. & Whitehead, A. S. 1146 (Prot.)
Holemans, K., with Wanson, 826 (Hel.)
Holland, 766 *bis* (B.R.)
Holland, G. P., (93) (Ent.)
Holmes, E. G., Gee, F. L., Asea, S. B., Bhima, S., Chagula, W. K., Evelia, T. R., Kasirye, J. W. S., Kiboneka, G. H., Lugazia, M. Y., Majale, M., Mughairy, M. S., Ochieng, E. C., Gesa, J. I. H. & Magambo, J. C., 397 (Haem.)
Holmes, R. H., with Enos, 927 (Misc. Dis.)
Holstein, M., 9 (Mal.)
Holt, C. J., with Jewett & Burner, 902 (Hel.)
Hoogana, L., with Kaplan & Zuelzer, (190), 190, 191 (Haem.)
Hoogstraal, H., Huff, C. G. & Lawless, D. K., 446 (Mal.)
Hopewell, W. W., with Arnason, Brown, Fredeen & Rempel, (92) (Ent.)
Hopkins, G. H. E., 460 (Pl.)
Hora, S. L., with Pandit, 1106 (Chl.)
Horgan, E. S., 935 (Reports, etc.)
Höring, F. O., 602 (B.R.)
Hormann, H., 234 (Mal.)
Hornabrook, R. W., 921 (Vms.)
Horne, G. O. & Mole, R. H., 79, 923 (Heat Str.)
Horrenberger, R., 354 (Typh.)
—— & Roubert, L., 353 (Typh.)
Horsman, R. K., with Hentel & Newton, 671 (Der.)
Hortling, H., with Telkkä, Putkonen & Teir, 740 (Lep.)
Horton, S. H., Jr. 277 (Hel.)
Horvath, S. M., with Eichna, Park, Nelson & Palmes, 403 (Heat Str.)
Hosoya, H., with Sasa, Kano, Hayashi, Kimura, Miura, Oyama, Sato & Hasegawa, 588 (Ent.)
Hotovy, R., with Eichholtz, 187 (Hel.)
Howell, D. E., with Laake, Dahm, Stone & Cuff, (590) (Ent.)
Hrenoff, A. K., 894 (Am.)
—— & Nakamura, M., 992 (Am.)
Hsiao, T. & Bohart, R. M., 588 (Ent.)
Hsü, H. F., with Li, (567) (Hel.)
Hsu, P., with Kung, 391 (Def. Dis.)
Huang, T. C., with Chow, 930 (Ent.)
——, with —— & Yul, 759 (Ent.)
Huard, P., 467 (Am.), 747 (Hel.)
—— & Tran Anh, 666, 1030 (Hel.)
Hudemann, H. & Mücke, D., 558, 812 (R.F.)
Huebner, R. J. & Bell, J. A., 545 (Typh.)
——, with Luoto & Stoenner, 546 (Typh.)
——, with Ransom, 545 (Typh.)
Hueck, O., 741 (Lep.), 1020 (Hel.)
Huff, C. G., 525 (Mal.)
——, with Hoogstraal & Lawless, 446 (Mal.)
——, Marchbank, D. F., Saroff, A. H., Scrimshaw, P. W. & Scrimshaw, P. W. & Shiroishi, T., 446 (Mal.)
Hughes, J. H. with Porter, (205) (Ent.)
Hughes, M. H., with Colbourne, Edington & Ward-Brew, 685 (Reports, etc.)
—— & Sarkies, J. W. R., 1031 (Hel.)
Hughes, T. E., 181 (Hel.)
Hughes, T. P. & Perlowagora, A., 547 (Y.F.)
Hugonot, R., 510 (Mal.)
Huhtala, A., 572 (Hel.)
Hulshoff, A. A., 712 (Mal.)
Humes, A. G., 382 (Hel.)
Le Van Hung, 706 (Mal.)
Hunninen, A. V., Young, M. D. & Burgess, R. W., 336 (Mal.)
Hunter, G. W., Dillahunt, J. A., Faust, E. C., Modic, J. L., Connellan, J. D., Bennett, H. J. & Ingalls, J. W., Jr., 570 (Hel.)
——, with Pan, 1012 (Hel.)
——, with —— & Kaufman, 1019 (Hel.)
Huntington, R. W., Jr., Eichold, S. & Scott, O. K., 488 (Hel.)
Husman, C. N., with Blanton, Travis & Smith, 91 (Ent.)
——, with McDuffie, Eddy & Clark, 415 (Ent.)
Hutchinson, M. P., with Duggan, 957 (Tryp.)
Huttel, V., (433) (Mal.)
Huttel, W., (217) (Reports, etc.)
——, with Harant, 70 (Vms.)
Hyman, G. A., with Conan, 497 (Der.)

I

Ibarra, L. M., with Sepulveda, 739 (Ys.)
Ibarra, R., with Pardo-Castello, Trespalacios, Tiant, Jose Mestre & Beade, 652 (Lep.)
Iboleón Hurtado, E., (1135) (Hel.)
Ibrahim, K. A., with Donegani, Ikin & Mourant, (280) (Haem.)
Ibuka, K., with Sato, Tadokoro & Kurihara, 987 (Chl.)
d'Ignazio, C., 246 (Typh.), 473 (Lep.), 819, 821 (Hel.)
—— & Benetazzo, B., 353 (Typh.)
—— & Codeleoncini, E., 139 (Typh.)
——, with de Francesco, 949 (Mal.)
—— & Rosa, B., 35 (Rab.)
—— & Termine, L., 353 (Typh.), (365) (Am.)
Ikegami, M., with Yaoi & Yoshino, 629 (Pl.)
Ikin, E. W., with Donegani, Ibrahim & Mourant, (280) (Haem.)
Imboden, C. A., Jr., Cooper, W. C., Coatney, G.R. & Jeffery, G. M., 523 (Mal.)
——, with ——, —— & ——, 439 (Mal.)
Incho, H. H., with Jones & Schroeder, 211 (Ent.)
Inclán Bolado, J. L., with Vara López, (902) (Hel.)
Indian Research Fund Association, 252 (Pl.), 438 (Mal.), (473) (Lep.)
Information, Central Office of: Empire Information Services, 791 (Tryp.)
Ingalls, J. W., Jr., with Hunter, Dillahunt, Faust, Modic, Connellan & Bennett, 570 (Hel.)
Ingbar, S. H. & Kass, E. H., 919 (Haem.)
Innes, J. R., 160, (369), 563, 643 *bis*, 995 (Lep.)
Inoki, S., Nagai, A., Kitamura, T., Takada, S. & Nakabayashi, T., 633 (Am.)
Intengan, C. L., with Burch, Salcedo, Carrasco & Caldwell, 64 (Def. Dis.)
Introzzi, P., 1135 (Hel.)
Iriarte, D. R., (204) (Ent.)
Irvine, C., 1129 (Lep.)
Isenberg, H. J., with Augustine, 380 (Hel.)
Ishihara, S. J., with Felsenfeld, Soman, Waters & Norsen, 988 (Chl.)
——, with ——, ——, Young, Yoshimura & Waters, 987 (Chl.)
——, with —— & Young, 258 (Chl.)
Ishii, N., with Asano, Kameda, Tamemasa & Chida, 1086 (Mal.)

Ishii, Y., 40 (Am.)
Ishino, K., 362, 631 *bis* (Am.)
Israels, S. & Shuman, B., 205 (Ent.)
Itano, H. A., with Wells, 579 (Haem.)
Ito, J., with Komiya, (1133) (Hel.)

J

Jachowski, L. A., Jr., Otto, G. F. & Wharton, J. D., 824 (Hel.)
Jackson, C. H. N., 133 (Tryp.)
——, with Burtt, 530 (Tryp.)
Jackson, E. B. & Smadel, J. E., 795, 978 (Typh.)
——, with —— & Ley, 978 (Typh.)
Jacob, V. P., with Kalra & Rao, 811 (R.F.)
Jacobs, E. C., Butz, W. C. & Felts, J. H., 585 (Misc. Dis.)
Jacobs, L., 632 (Am.)
Jacobson, J. N. & Madden, P. C. W., (466) (Am.)
Jacono, G. & Colucci, C. F., 166 (Lep.)
Jadin, J., 879 (Typh.)
—— & Giroud, P., 543, 885 (Typh.)
——, with ——, 544, 545, 727 (Typh.)
——, with —— & Jezierski, 1101 (Typh.)
Jagadisan, T. N., (48) (Lep.)
Jahan, P., with Canet, 180 (Hel.)
Jalili, M. A., Nakkash, B. Y. & Taj-el-Din, H. M., 751 (Haem.)
Jamil, A. K., with Watson & Pringle, 1005 (Hel.)
Janicki, M., Konopacka, B. & Dymowska, Z., 900 (Hel.)
Janssen, E. & le Roux, J. S., 62 (Def. Dis.)
Janssens, P. G., 399 (Haem.)
——, Verstraete, N. & Sieniawski, J., 864 (Mal.)
Jánszky, B., (69) (Vms.)
Jany, E., 985 (Pl.)
Jaramillo-Arango, J., 304 (B.R.)
Jarpa, G., A., with Agosín, Christen & Atías, 720 (Tryp.)
——, with Alba, 749 (Hel.)
——, Christen, R., Agosin, M. & Pizzi, T., 1096 (Tryp.)
Jasper, D. E., (408) (Prot.)
Jaswant Singh, 1067 (Mal.)
—— & Nair, C. P., 1063 (Mal.)
——, Raghavan, N. G. S., Nair, C. P. & Basu, P. C., 713 (Mal.)
——, Ramakrishnan, S. P. & David, A., 878 (Tryp.)
——, Ray, A. P. & Nair, C. P., 20 (Mal.), 94 (Lab.), 334 (Mal.)
Jaujou, C., Camain, R. & Adam, J. P., (208) (Ent.)
Jausion, H., Calop, R., Benard, P. & Aboud, B., (837) (Heat Str.)
Jaworska-Kozakowa, K., 672 (Oph.)
Jean, L., with Payne & Bellerive, 1118 (Ys.)
Jeffery, G. M., 184 (Hel.)
——, with Cooper, Coatney & Imboden, 439 (Mal.)
——, with Edgcomb, Arnold, Yount, Alving, Eichelberger, Eyles & Young, 440 (Mal.)
——, Eyles, D. E. & Young, M. D., 704 (Mal.)
——, with Imboden, Cooper & Coatney, 523 (Mal.)
Jelliffe, D. B., 279, 1026 (Hel.), 472 (Ys.), (809) (Am.), 875 (Bl.), 898 (Lep.), 1140 (Def. Dis.), 1148 (Prot.)
Jellison, W. L. & Gregson, J. D., 86 (Misc. Dis.)
Jenkins, D. W. & Hassett, C. C., (928) (Ent.)
Jensen, J. A. & Fay, R. W., 1153 (Ent.)
——, Sumerford, W. T. & Fay, R. W., 416 (Ent.)
Jervis, O. & Montalvan C., J. A., 346 (Tryp.)
Jettmar, H. M., 432 (Mal.)
—— & Anschau, M., 1153 (Ent.)
—— & Exner, H., 905 (Hel.)
Jewett, J. S., Burner, A. M. & Holt, C. J., 902 (Hel.)
Jezierski, A., with Giroud & Jadin, 1101 (Typh.)
Jíra, J., with Kučera, 810 (Am.)
Joaquin Canabal, E., with Piaggio Blanco, Dubourdieu, Dighiero & Grosso, (56) (Hel.)
Lie Kian Joe & Bras, G., 56, 172 (Hel.)
Johansen, F. A. & Erickson, P. T., 474 (Lep.)
John, W. C., with Schwartz & Mansuy, 437 (Mal.)
Johnson, A. H., with Gartrell, 129 (Mal.)
Johnson, B. A., with Meleney (756) (Der.)
Johnson, C. G., 203 (Ent.)
Jonchère, H., 717 (Tryp.)
——, with Dejou, Koerber, Labail & d'Almeida, 666 (Hel.)
Jones, B. M., 27 (Typh.)
Jones, H. A., Schroeder, H. O. & Incho, H. H., 211 (Ent.)
Jones, T. W. T., 949 (Mal.)
de Jong, J. R., (209) (Ent.)
Jose, F. R., with Salcedo, Bamba, Carrasco, Chan, Concepcion, deLeon, Oliveros, Pascual, Santiago & Valenzuela, 494 (Def. Dis.)
Jose Mestre, J., with Pardo-Castello, Trespalacios, Tiant, Ibarra & Beade, 652 (Lep.)
José Osimani, J., Veríssimo, S. & Baycé Carbonell, P., 347 (Tryp.)
Joseph, A., with Loughlin & Schaeffer, 560, 562 (Ys.)
——, with Schaeffer & Loughlin, 562 (Ys.)
Josephson, E. S., with Greenberg & Taylor, 873 (Mal.)
——, Taylor, D. J., Greenberg, J. & Ray, A. P., 862 (Mal.)
——, with ——, —— & ——, 873 (Mal.)
Joslin, B. S., with Bauer, Parker, Hall, Benson, Hightower, Snyder, Venable & Woodward, 979 (Typh.)
J. Amer. Med. Ass., (594) (Ent.)
J. National Malaria Soc., 1078 (Mal.)
J. Parasitology, 418 (Reports, etc.)
J. Philippine Med. Ass., 1018 (Hel.)
Joyeux, C., Baer, J. G. & Gaud, J., 173 (Hel.)
—— & Sicé, A., 302 (B.R.)
Joyner, L. P., with Fulton, King, Osbond & Wright, 365 (Am.)
——, with —— & Price, 734 (Am.)
Juárez Juárez, E., with Lozano Morales, 787 (Mal.)
Judson, W. E., 886 (Den.)
Jullien-Vieroz, R., Caubet, P. & Montestruc, E., 52 (Hel.)
Justesen, V. H. & Koolsbergen, N., 119 (Mal.)

K

Kaether, H. & Becker, H. W., 795 (Typh.)
Kahn, R. L., 997 (Lep.)
Kalra, S. L., Jacob, V. P. & Rao, K. N. A., 811 (R.F.)
Kameda, Y., with Asano, Tamemasa, Ishii & Chida, 1086 (Mal.)

Kano, R., with Sasa & Hayashi, (293) (Ent.)
——, with ——, ——, Kimura, Miura, Oyama, Sato, Hosoya, H. & Hasegawa, 588 (Ent.)
——, with —— & Takahasi, (293) (Ent.)
Kant, L., with Rudra, 198 (Misc. Dis.)
Kantarjian, A. D., with Al-Witry & Shaby, 750 (Def. Dis.)
Kaplan, E. & Zuelzer, W. W., with Hoogana, L., (190), 190, 191 (Haem.)
Kaplan, S., with Carlos Radice, 581 (Der.)
Kapur, P. L., with Dharmendra, Dey & Bose, 374 (Lep.)
Karlson, A. G., with Feldman & Grindlay, 1120 (Lep.)
Karode, N. R., (747) (Hel.)
Kasirye, J. W. S., with Holmes, Gee, Asea, Bhima, Chagula, Evelia, Kiboneka, Lugazia, Majale, Mughairy, Ochieng, Gesa & Magambo, 397 (Haem.)
Kasper, J. A., Cope, E. J., Lyon, M. & White, M., 361 (Am.)
Kass, E. H., with Ingbar, 919 (Haem.)
——, Neva, F. A. & Finland, M., 725 (Typh.)
Kassel, R. & Melnitsky, I., (891) (Am.)
Kaufman, E. H., with Pan & Hunter, 1019 (Hel.)
Kausche, G. A., with Hengel & Sheris, 249 (Typh.)
Kazzaz, D. S. & Tabbara, R., 1132 (Hel.)
Keaty, C., with Lamb, Shelmire, Cooper & Morgan, 71 (Der.)
Keegan, H. L., Blauw, A. S. & Anderson, R. I., 401 (Vms.)
Keener, G. G., with Cutkomp & Hess, 297 (Ent.)
Kehl, R., with Werle & Koebke, (580) (Vms.)
Keil, H., with Rotberg & Bechelli, 266 (Lep.)
Keiser, F., 89 (Ent.)
Keizer, D. P. R., 578 (Hel.)
Keller, P., 487 (Hel.)
—— & Hide, C. G., 824 (Hel.)
Kellersberger, E. R., (995) (Lep.)
Kelley, M. J., with Schubert, 432 (Mal.)
Kelso, R. W., Jr., with Stempien & Marcus, 635 (Am.)
Kemp, H. & de Aguiar, H. A., 1157 (Ent.)
Kempson, D. A., with Chitty, 252 (Pl.)
Kenawy, M. R., 51 (Hel.)
Kendall, S. B., 172 (Hel.)
—— & McCullough, F. S., 1020 (Hel.)
Kennaway, E., 845 (B.R.)
Kenney, M. & Hewitt, R., 488 (Hel.)
Kepp, R. McF., with Smith, Saito, Beard, Clark & Eddie, 73 (Der.)
Kerharo, J., with Bouquet, (390) (Def. Dis.)
Kermack, W. O. & Storey, N. E., (864) (Mal.)
Kernevez, H. & Chassain, J., 959 (Tryp.)
Kerr, D. E., with Dolman, Chang & Shearer, 642, 994 (R.F.)
Kershaw, W. E., 177, 574 (Hel.)
Keskar, A. Y. & Bokil, K. V., (566) (Lep.)
Kessel, J. F., with Kritzer & Biddle, 284 (Der.)
Ketelaar, J. A. A. & Hellingman, J. E., (763) (Ent.)
Kettle, D. S., (1152) (Ent.)
Keys, A., Brožek, J., Henschel, A., Mickelsen, O. & Taylor, H. L., with Simonson, E., Skinner, A. S. & Wells, S. M., 767 (B.R.)
Keys, D. N., 788 (Mal.)
Khalaf, G., with Halawani, Latif & Taha, 1013 (Hel.)
Khan, N., 354 (Typh.), 1067 (Mal.)
Khlentzos, C. T., 199 (Misc. Dis.)
Kiboneka, G. H., with Holmes, Gee, Asea, Bhima, Chagula, Evelia, Kasirye, Lugazia, Majale, Mughairy, Ochieng, Gesa & Magambo, 397 (Haem.)
Kikuth, W., (12) (Mal.)
—— & Mudrow-Reichenow, L., 271 (Lep.)
Kimura, M., with Sasa, Kano, Hayashi, Miura, Oyama, Sato, Hosoya & Hasegawa, 588 (Ent.)
Kinawy, M., with Fahmy, (753) (Haem.)
Kincaid, R. K., with Robinson, Gerking & Turrell, 78 (Heat Str.)
——, with —— & Rhamy, 196 (Heat Str.)
Kinder, E., with Kleinschmidt, 622 (Tryp.)
King, C. G., with Drummond, Wilder & Williams, 767 (B.R.)
King, H., with Fulton, Joyner, Osbond & Wright, 365 (Am.)
King, W. V., 413 (Ent.)
Kirchberg, E., 1152 (Ent.)
Kirk, R., with Lewis, 534 (Leish.)
Kirsch, E., & Westphal, A., 717 (Tryp.)
Kirwan, E. O'G., Langrishe, J. duP., Martin, G. E. & Pollock, G. A., 939 (B.R.)
Kitamura, O., with Arakawa, 499 (Oph.)
Kitamura, T., with Inoki, Nagai, Takada & Nakabayashi, 633 (Am.)
Kitaoka, M. & Shishido, A., 624, 973 (Typh.)
Kitchen, D. K., with Rein & Pétrus, 158 (Ys.)
Kivits, M., 896 (Ys.)
—— & Friedheim, E. A. H., 896 (Ys.)
Klausová, L., with Pokorny, 177 (Hel.)
Kleeberg, F. & Birnbaum, D., 265 (Am.)
Kleeberg, J. & Birnbaum, D., 263 (Am.)
Klein, W. E. & Bradshaw, R. H., 670 (Vms.)
Kleine, F. K., 134 (Tryp.)
Kleinot, S., 466 (Am.)
Kleinschmidt, A., 1090 (Tryp.)
—— & Kinder, E., 622 (Tryp.)
Klenerman, P., 189 (Def. Dis.), 1148 (Prot.)
Klosa, J., 62 (Hel.)
Kluttz, J., with Mackie, Simpson & Tuttle, 857 (Mal.)
Kneeland, Y., Jr., with Rose & Gibson, 547 (Typh.)
Knipe, F. W., 781 (Mal.)
Knutsen, B., 701 (Mal.)
Knüttgen, H., with Fuhrmann, 117 (Mal.)
Koch, K. R. & Kux, P., 1132 (Hel.)
Koch, W. & Steinitz, H., 1130 (Hel.)
Koch-Weser, D., with Eichbaum & Leao, 1023 (Hel.)
Kodijat, R., with Hill & Sardadi, 1117 (Ys.)
Koebke, K., with Werle & Kehl, (580) (Vms.)
Koenigstein, R. P., Chiu, Y. S. & Tsai, K. C., 533 (Leish.)
Koerber, R., 793 (Tryp.)
——, with Dejou, Jonchère, Labail & d'Almeida, 666 (Hel.)
Kohn, J., 464 (Am.)
Komiya, Y. & Ito, J., (1133) (Hel.)
Kondi, A., with Foy, 528 (Bl.)
——, with —— & Alexandrides, 918 (Haem.)
——, with —— & Brass, 918 (Haem.)
——, with ——, Hargreaves & Lowry, 67 (Haem.)

Kondo, A., with Nakano, Yaoi & Nakagami, 459 (Rab.)
——, Yaoi, H., Nakagami, S. & Nakano, M., 459 (Rab.)
——, with ——, —— & ——, 34, 459 (Rab.)
Konopacka, B., with Janicki & Dymowska, 900 (Hel.)
Konstantinidu, S., with Fakaçelli, 927 (Misc. Dis.)
Koo, K., with Liu & Ch'en, (1038) (Haem.)
Koolsbergen, N., with Justesen, 119 (Mal.)
El Kordy, M. I., with Halawani & Abdallah, 155, 806 (Am.)
Koshy, P., 152 (Am.)
Kostermans, D., (45) (Lep.)
Kostich, D. Y., 969 (Leish.)
Kostrzewski, J., (886) (Typh.)
Koumans, A. K. J., 150 (Am.)
Kouwenaar, W., van Steenis, P. B. & Winckel, Ch. W. F., 938 (B. R.)
Krishnan, K. V., 546 (Typh.)
Kritzer, M. D., Biddle, M. & Kessel, J. F., 284 (Der.)
Krusé, C. W., Philen, E. A. & Ludvik, G. F., 615 (Mal.)
Kryński, S., 724 (Typh.)
—— & Woyciechowska, S., 723 (Typh.)
Kshatriya, K. C., Patel, S. R. & Nargund, K. S. (523) (Mal.)
Kučera, K. & Jíra, J., 810 (Am.)
Kuhns, D. M., with Shively, 162 (Lep.)
Kuitert, L. C. & Hitchcock, J. D., 513 (Mal.)
Kuitunen-Ekbaum, E., with Brown, Green & Boag, 475 (Hel.)
Kumagai, A., 974 (Typh.)
Kumm, H. W., 31 (Y.F.)
—— & Laemmert, H. W., Jr., 455, 456 (Y.F.)
——, with —— 456 (Y.F.)
Kung, L. & Hsu, P., 391 (Def. Dis.)
Kuntz, R. E. & Stirewalt, M. A., 273 (Hel.)
Kupka, E. & Anschau, K., 229 (Mal.)
Kurihara, T., with Sato, Tadokoro & Ibuka, 987 (Chl.)
Kux, P., with Koch, 1132 (Hel.)
Van der Kuyp, E., 109, 437 (Mal.), (387) (Hel.)

L

Laake, E. W., Howell, D. E., Dahm, P. A., Stone, P. C. & Cuff, R. L. (590) (Ent.)
Labail, P., with Dejou, Jonchère, Koerber & d'Almeida, 666 (Hel.)
LaCasse, W. J., 90 (Ent.)
Lacaz, C. da S., 1144 (Der.)
Lacerda, M. T., with Fonseca, Pinto, Colaço, Oliveira, Branco, da Gama & Franco, 980 (Typh.)
Lacerda, N., with de Bustamante, Paracampos & Santos, 319 (Mal.)
Lackman, D. B., with Parker, Pickens, Bell & Thraikill, 796 (Typh.)
Lacorte, J. G., 923 (Oph.)
Ladell, W. S. S., 836 (Heat Str.)
Ladkin, R. G., with Raper, 196 (Misc. Dis.)
Laemmert, H. W., Jr. & Kumm, H. W., 456 (Y.F.)
——, with ——, 455, 456 (Y.F.)
Lagarde, J., with Le Gac & Mulet, 496 (Der.)
Lagrange, E., 489, 902, 1013 (Hel.)
—— & Scheecqmans, G., 571, 1011, 1012 (Hel.)
——, —— & Sarkissian, M., 1013 (Hel.)
Lahiri, S. C., 551 (Chl.)
Laird, M., 322, 790 (Mal.)
Laird, R. L., Dubin, I. N. & Drinnon, V. P., 335 (Mal.)
——, with —— & ——, 334 (Mal.)
Lal, S. B., 833 (Ep. Dropsy), 1138 (Def. Dis.)
Lalouel, J., 504 (Reports, etc.)
Lama, A., 805 (Rab.)
Lamb, J. H., Shelmire, B., Cooper, Z., Morgan, R. J. & Keaty, C., 71 (Der.)
Lambotte-Legrand, C., with Lambotte-Legrand, 399, 918 (Haem.)
Lambotte-Legrand, J. & Lambotte-Legrand, C., 399, 918 (Haem.)
Lamy, L., with Deschiens, 630 (Am.)
—— & Roux, H., 265 (Am.)
Lancaster, H. O., 463 (Am.)
Landfvar, A. F., (822) (Hel.)
Landquist, J. K., (713) (Mal.)
Lane, J., (401) (Der.), 588 (Ent.)
Lang, F., (56) (Hel.)
Lange, R. D., Minnich, V. & Moore, C. V., 832 (Haem.)
Langmuir, A. D., with Andrews & Quinby, 430 (Mal.)
Langrishe, J., duP., with Kirwan, Martin & Pollock, 939 (B.R.)
Languillon, J., 608 (Mal.), 751, (832) (Haem.), (878) (Tryp.), 1127 (Lep.)
——, with Mauzé, 171 (Hel.)
Lanz, P., with Chabaud, 1137 (Hel.)
Lapeyssonnie, L., 792, 959 (Tryp.)
Lapidus, M., (1131) (Hel.)
Lapierre, J., with Galliard, 129, 869 (Mal.)
Lapieza Cabral, P., with Borzone, Alvarez de Toledo & Aizemberg, 349 (Tryp.)
Larghero Ybarz, P., (274) (Hel.)
Larizza, P. & Ventura, S., 1135 (Hel.)
Laskowski, L., Stanton, M. F. & Pinkerton, H., 730 (Bart.)
——, with —— & ——, 143 (Bart.)
de la Lastra Soubrier, J. M., 1119 (Lep.)
Laszlo, H. P., 451 (Tryp.)
Latif, N., with Halawani, Taha & Khalaf, 1013 (Hel.)
Latimer, F. R., Webster, J. E. & Gurdjian, E. S., 628 (Rab.)
Lauderdale, R. W., with Morrison, Crowell & Mote, (590) (Ent.)
Launoy, L., 342 (Tryp.)
Laur, A. & Rabenschlag, K., 729 (Typh.)
Laurent, G., with Giroud, Le Gac & Brizard, 626, 980 (Typh.)
Laurent, J., with Gaud, 512 (Mal.)
Lauret, L., with Laviron, 648, 741, 1123 (Lep.)
——, with —— & Schneider, 1124 (Lep.)
——, with Schneider, Laviron & Basset, 651 (Lep.)
Laurie, W., 1027 (Hel.)
Laven, H., 110 (Mal.)
—— & Westphal, A., 201 (Prot.)
Lavergne, J., 1060 (Mal.)
Laviron, P. & Lauret, L., 648, 741, 1123 (Lep.)
——, —— & Schneider, J., 1124 (Lep.)
——, with Schneider, Lauret & Basset, 651 (Lep.)

Lawless, D. K., with Hoogstraal & Huff, 446 (Mal.)
Lawlis, J. F., Jr., with McCowen & Callender, 640 (Am.)
Lawrence, J. J., with Bearup, 301 (Ent.)
Leao, A. T., with Eichbaum & Koch-Weser, 1023 (Hel.)
Leavell, B. S., with Shotton & Crockett, 753 (Haem.)
Lecuiller, A., with Floch & Destombes, 742, 999 *bis* (Lep.)
Ledo, J., with Deane, Freire & Tabosa, 19 (Mal.)
Lee, D. H. K., 287 (Heat Str.)
Leeson, F., (193) (Vms.)
Leeson, H. S., 103 (B.R.), 1156 (Ent.)
Lefebvre, E., with Soulage, Farinaud & Tauzin, 253 (Pl.)
Lefrou, G., 919 (Vms.)
Legeay, G., with Guerret, (145) (Rab.)
Legler, F., 573, 663 (Hel.)
Legros, A., with Devignat & Vercammen-Grandjean, 993 (Am.)
Lehman, A. J., Hartzell, A. & Ward, J. C., (594) (Ent.)
Lehmann, H., 763 (Reports, etc.)
Leitch, N., 763 (Reports, etc.)
Leleup, N., with Vincke, (760) (Ent.)
Lelong, M., Le Tan Vinh, R. J., Desmonts & Dupré-Bouteloup, L., 1147 (Prot.)
Lemierre, 217 (B.R.)
Lendle, L. & Schneider, H.-H., 912 (Hel.)
Lenelle, J. & Parent, M., 1138 (Def. Dis.)
Lennette, E. H. & Clark, W. H., 543 (Typh.)
——, with —— & Meiklejohn, 626 (Typh.)
——, with DeLay & DeOme, 140 (Typh.)
de Leon, D. M., with Garduño, 1128 (Lep.)
deLeon, J. F., with Salcedo, Bamba, Carrasco, Chan, Concepcion, Jose, Oliveros, Pascual, Santiago & Valenzuela, 494 (Def. Dis.)
de Leon, J. R., 532 *bis* (Tryp.)
León, L. A., 1097 (Leish.)
Leonardi, G., with de Muro, 752 (Haem.)
León-Blanco, F., with Angulo, Watson, Wedderburn & Varela, 1117 (Ys.)
Leonhardt, H., 151 (Am.)
Lépine, P. & Croissant, O., 1104 (Rab.)
——, Pavilanis, V. & Atanasiu, P., 887 (Rab.)
Leprat, J., with Brouet & Marche, 657 (Hel.)
Leprosy in India, (160), 740 (Lep.)
le Roux, J. S., with Janssen, 62 (Def. Dis.)
leRoux, P. L., 177, 1006 (Hel.)
Lester, H. M. O., 951 (Tryp.)
Levaditi, C., 858 (Mal.)
—— & Vaisman, A., 445 (Mal.)
——, —— & Chaigneau, H., 739 (R.F.)
Levi-Castillo, R., (547), (803), 981 (Y.F.)
Levin, S., with Westwood, 759 (Misc. Dis.)
Levin, W. C., with Schneider, 192 (Haem.)
Levina, D. A., 658 (Hel.)
Levine, H. B. & Garber, E. D., 145 (Pl.)
Levine, M., 646 (Lep.)
Levinson, Z., with Ascher & Reuter, 1155 (Ent.)
Lévy, G., with Farinaud & Frézières, 247 (Typh.)
——, with —— & Gros, 674 (Misc. Dis.)
——, with de Rothschild, 709 (Mal.)
Lewis, D. J., 24 (Tryp.)
—— & Kirk, R., 534 (Leish.)
Lewis, E. A., 449, 951 (Tryp.)
Lewis, J. L., Jr., with Terry & Sessoms, 535 (Leish.)
Lewis, R., with Sadun, Ané & Fuller, 261 (Am.)
Lewis, R. A., with Faust, Sadun, Everritt & Bradin, 148 (Am.)
Ley, H. L., Jr., with Smadel, Diercks & Traub, 355 (Typh.)
——, with ——, ——, ——, Tipton & Frick, 977 (Typh.)
——, with —— & Jackson, 978 (Typh.)
Li, S. Y. & Hsü, H. F., (567) (Hel.)
Liang, K. C., with Paul & Watson, 443 (Mal.)
Lichtenstein, B. W. & Simon, A., 657 (Hel.)
Liebmann, H., 37 (Am.)
Lima, F. O., with Correa & Coda, 114 (Mal.)
Lima, J. F., with Vasconcellos, 167 (Hel.)
Lima, M. M., with Rachou & Ferreira, 618 (Mal.)
Lindén, V., with Böe & Tveit, 754 (Der.)
Lindquist, A. W., Roth, A. R., Yates, W. W., Hoffman, R. A. & Butts, J. S., 931 (Ent.)
——, with Yates, Gjullin & Butts, (1049) (Ent.)
Lindsay, D. R. & Haines, T. W., 1050 (Ent.)
Linhard, J., 1037 (Haem.)
Link, V. B., 146, 548, 1105 (Pl.)
Lins, S. A., Guimarães, F. N., Vasconcellos, P. & Miranda, J. L., 160 (Ys.)
Lints, H. A., with Coatney, Cooper, Eyles, Culwell & White, 124 (Mal.)
Lipparoni, E., 477 (Hel.), 578 (Def. Dis.), 810 (R.F.), 866 (Mal.)
Lippi, M., 44, 162 (Lep.), 55 (Hel.), 233 (Mal.)
—— & Floris, M., 399 (Haem.)
—— & Tripodi, P., 242 (Leish.)
Lips, M., with Vincke, 714 (Mal.)
Liquori, A. M., 830 (Haem.)
——, with Perutz & Eirich, (1142) (Haem.)
Lisa, J. R., with Seife, 168 (Hel.)
Lister, L. M., with Parker, Bauer, Woodward & Hall, 540 (Typh.)
Liu, C., Ch'en, W. & Koo, K., (1038) (Haem.)
Liu, F., with Chang, 971 (Leish.)
——, with ——, Zia & Chao, 729 (Typh.)
Lloyd, O. C., with Mulligan & Sommerville, 21 (Mal.)
Lock, J. A., 344 (Tryp.)
Lodens, J. G., with van Goor & Gomez, 325 (Mal.)
Loe, F., with Pijoan & Payne, 1043 (Oph.)
Loewenthal, L. J. A., 406 (Misc. Dis.)
Lofgren, R., 452 (Leish.)
Lombard, G. & Marchalant, L., 133 (Bl.)
Lommel, F., 904 (Hel.)
London School of Hygiene and Tropical Medicine, Memoir No. 7, 103 (B.R.)
Longanecker, D. S., 1115 (R.F.)
Lopez-Sanabria, U., with Rodriguez-Molina, Acevedo, Torres & Ramirez-Rodriguez, 481 (Hel.)
Lopez-Toca, R., with Spies, Garcia Lopez, Milanes & Reboredo, 400 (Haem.)
——, with ——, ——, ——, —— & Stone, 399 (Haem.)
Löpmann, A., with Eichler & Pagast, 1064 (Mal.)
Lord, K. A. & Potter, C., (933) (Ent.)
Lotte, A. J., with Raynal, 960 (Tryp.)
Loughlin, E. H., Joseph, A. & Schaeffer, K., 560, 562 (Ys.)
——, with Schaeffer & Joseph, 562 (Ys.)

Lourenço Marques, Moçambique, 791 (Tryp.)
Lourie, E. M., 763 (Reports, etc.)
Loveday, P. M., with Winteringham & Harrison, 501 (Ent.)
Low, C., 177 (Hel.)
Lowe, J., 374, 649, 1000 (Lep.)
—— & Davey, T. F., 1124 (Lep.)
Lowry, J., with Foy, Kondi & Hargreaves, 67 (Haem.)
Lozano Morales, A., 123 (Mal.)
—— & Juárez Juárez, E., 787 (Mal.)
Lu, J., with Chung, Chang, Chang, Feng & Tsao, 242 (Leish.)
Lucchini, A., with Gajardo Tobar, Apablaza, Uribe, Benavides, Vargas, Cepeda, Rojas, Zeldis & De la Fuente, 381 (Hel.)
Lucena, D. T., (380) (Hel.)
de Lucena, D. T., 435, 863, (1084) (Mal.)
——, with Bequaert, 743 (Hel.)
Ludvik, G. F., 236 (Mal.)
——, with Krusé & Philen, 615 (Mal.)
——, Snow, W. E. & Hawkins, W. B., 616 (Mal.)
Lugazia, M. Y., with Holmes, Gee, Asea, Bhima, Chagula, Evelia, Kasirye, Kiboneka, Majale, Mughairy, Ochieng, Gesa & Magambo, 397 (Haem.)
Luis Carri, E., 366 (Am.)
Lumsden, W. H. R., 981 (Y.F.)
—— & Buxton, A. P., 1102 (Y.F.)
—— & Yofe, J., 103 (B.R.)
Luoto, L., Huebner, R. J. & Stoenner, H. G., 546 (Typh.)
Lurie, H. I. & de Meillon, B., 901 (Hel.)
Luttermoser, G. W., with Haskins & Brady, 41 (Am.)
Luyken, R., 1138 (Def. Dis.)
Lyman, F. E., with Bradley, 19 (Mal.)
Lyon, M., with Kasper, Cope & White, 361 (Am.)

M

Macan, T. T., 103 (B.R.), 231 (Mal.)
McArthur, J., 434, 1065 (Mal.)
Macaulay, J. W. & Shaw, G. D., (346) (Tryp.)
MacCallum, F. O., with Marmion, Rowlands, Thiel, McDonald & Phipps, 1100 (Typh.)
Maccarrone, A., with Buonomini & Motta-Diana, 230 (Mal.)
McCarten, W. G. & Griffin, A. M., (365) (Am.)
McConnachie, E. W., 1085 (Mal.)
——, with Bishop, 1085 (Mal.)
McConnell, K. P., with Watts, 571 (Hel.)
McCowen, M. C., Callender, M. E. & Lawlis, J. F., Jr., 640 (Am.)
McCoy, J. E., with Tsuchiya, 860 (Mal.)
McCullough, F. S., with Kendall, 1020 (Hel.)
Macdonald, A., 288 (Prot.)
Macdonald, G., 17 (Mal.), 763 (Reports, etc.)
McDonald, J. R., with Marmion, MacCallum, Rowlands, Thiel & Phipps, 1100 (Typh.)
McDuffie, W. C., with Deonier & Cain, 412 (Ent.)
——, Eddy, G. W., Clark, J. C. & Husman, C. N., 415 (Ent.)
——, with Hocking & Twinn, 208 (Ent.)
Macedo, D. G., with Guimarães, 283 (Der.)
McFadzean, J. A., 869 (Mal.)
McGhee, R. B., 619 (Mal.)
McGowan, D., 1090 (Tryp.)
MacGregor, R. B., 128 (Mal.)
McIlroy, M. B., with Cates, 1089 (Tryp.)
Macindoe, N. M., with Bolliger, 971 (Leish.)
McKenna, J., with Haworth, Powell & Woodward, (894) (Am.)
Mackerras, I. M., with Mackerras, (414) (Ent.)
Mackerras, M. J. & Mackerras, I. M., (414) (Ent.)
Mackie, T. T., Simpson, T. W. & Tuttle, R. L., with Kluttz, J., 857 (Mal.)
Mackinnon, J. E., Artagaveytia-Allende, R. C., Vinelli, H., Niño, F. L., Ferrada-Urzúa, L. V. Alonso, G. & Donoso, R., 582 (Der.)
—— & Gurri, J., 195 (Der.)
McLeod, M., with Ranta, 358 (Chl.)
MacLeod, R. C., 442 (Mal.)
McMahon, J., with van Someren, 181 (Hel.)
McMullen, D. B., 1019 (Hel.)
McNamee, R. W., 211 (Ent.)
Macpherson, R. K., 77 (Heat Str.)
McQuown, A. L., 183 (Hel.)
McRobert, G. R., 583 (Misc. Dis.)
Madden, P. C. W., with Jacobson, (466) (Am.)
Madramany, P., 816 (Lep.)
Madwar, S. & Zahar, A. R., 929 (Ent.)
Maegraith, B. G., 14 (Mal.)
——, with Gall, (601) (Reports, etc.)
de Magalhães, O., 29 *bis*, (248) *bis* (Typh.)
——, Café, B. & Rocha, A., 728 (Typh.)
——, Freire, S. A. & Rocha, A., (541) (Typh.)
—— & Rocha, A., 541 (Typh.)
Magambo, J. C., with Holmes, Gee, Asea, Bhima, Chagula, Evelia, Kasirye, Kiboneka, Lugazia, Majale, Mughairy, Ochieng & Gesa, 397 (Haem.)
Magath, T. B., 1023 (Hel.)
Magdelenat, P., with Garipuy, (990) (Am.)
de Magistris, L., 458 (Rab.)
Magnusson, J. H., 676 (Prot.)
—— & Wahlgren, F., 408 (Prot.)
Magy, H. I., Dahl, A. H. & Geib, A. F., 678 (Ent.)
Maia, C. da C., 377 (Hel.)
Maier, E. H., 1045 (Misc. Dis.)
Maillot, L., 133, 238 (Tryp.)
Mainzer, F., (393) (Def. Dis.), 653 (Hel.)
Majale, M., with Holmes, Gee, Asea, Bhima, Chagula, Evelia, Kasirye, Kiboneka, Lugazia, Mughairy, Ochieng, Gesa & Magambo, 397 (Haem.)
Makstenieks, O., with Verlinde, 928 (Prot.)
Malaret. P. S., 644 (Lep.)
Malaya, Federation of, 122, 128 (Mal.), 207 (Ent.)
Maldonado Capriles, J., with Rehn & Henderson, 113 (Mal.)
Maldonado, J. F., Acosta Matienzo, J. & Vélez Herrera, F., 50, 1008 (Hel.)
Malfatti, M. G., 1119 (Lep.)
Mallet, R., (25) (Tryp.)
Mallick, K. C. B., with Sen Gupta & Bhattacharya, 594 (Lab.)
Manaland, C., (369) (Lep.)
Manninger, R., (983) (Rab.)
Manrique I., J., (1094) (Tryp.)
Manson, R. C., with Conrad & Peterkin, 623 (Leish.)
Manson-Bahr, P., 177 (Hel.), 686 (Reports, etc.)
Manson-Bahr, P. E. C., 829 (Def. Dis.), 830 (Haem.)
Mansuy, M. M., with Schwartz & John, 437 (Mal.)

Manwell, R. D., 875 (Mal.)
—— & Brody, G., 335 (Mal.)
Mao, W. S., 756 (Oph.)
Mara, L., 326 (Mal.)
March, R. B., with Metcalf, (298) (Ent.)
Marchalant, L., (764) (Reports, etc.)
——, with Lombard, 133 (Bl.)
Marchand, J. F., with Abrams & Hamblin, (594) (Ent.)
Marchbank, D. F., with Huff, Saroff, Scrimshaw & Shiroishi, 446 (Mal.)
Marche, J., with Brouet & Leprat, 657 (Hel.)
Marcus, D. M., with Stempien & Kelso, 635 (Am.)
Maretić, Z., 921 (Vms.)
Margolies, M. P., 579 (Haem.)
——, with Hanno, 398 (Haem.)
Marie-Suzanne, with Noël, 652 (Lep.)
Mariotta, D. A., with Oliver González, Biaggi, Acevedo, Hernández Morales, Santiago Stevenson & Pérez Santiago, 54 (Hel.)
Markos, B. G., with Peters, Thurman & Mulhern, 91 (Ent.)
Marmion, B. P., (542) (Typh.)
——, MacCallum, F. O., Rowlands, A. & Thiel, C. C., with McDonald, J. R. & Phipps, P. H., 1100 (Typh.)
—— & Stoker, M. G. P., 248 (Typh.)
Marples, M. J., 581 (Der.)
Martignolles, J. & Adjovi, P., (346) (Tryp.)
Martin, with Fontan, Verger, Bentéveat & Dubreuil, (367) (R.F.)
Martin, B., 601 (B.R.)
Martin, G. E., with Kirwan, Langrishe & Pollock, 939 (B.R.)
Martin, M., with Crosnier, Darbon, Ducourneau, Bouilloc & Ricossé, 991 (Am.)
Martinez, E., 972 (Leish.)
——, with Meyer, Suarez, Suarez & Benitez Gautier, 916 (Sp.)
Martinez, E. C., with Suarez, Suarez & Elias Latorre, 916 (Sp.)
Martinez Domínguez, V., 816 (Lep.)
Martinez Palacios, A., with Vargas, 506 (B.R.)
Martirani, I., & Azevedo, M. P., (835) (Vms.)
——, with ——, (835) (Vms.)
Marty, J., with Duroux & Tabusse (274) (Hel.)
——, with Germain, 969 (Leish.)
Maryon, M., with Shute, 702 (Mal.)
Massonnat, J., with Aubry, Boulard, Portier & Messerschmitt, (919) (Haem.)
——, with ——, —— & Zevaco, (902) (Hel.)
——, with Thiodet, Butori & Fourrier, 468 (Am.)
——, with ——, Pietri, Fourrier & Aubry, 992 (Am.)
Massouda, G., 1042 (Oph.)
Mastbaum, O., 1073 (Mal.)
Mathen, K. K., with Mukerji, 174 (Hel.)
Mathis, C., 456 (Y.F.)
Mathis, M., with Durand, 715 (Mal.)
Matilla, V., Aparicio Garrido, J., Diez Melchor, F. & Prieto Lorenzo, A., 176 (Hel.)
——, —— & Prieto Lorenzo, A., 573, 662 (Hel.)
——, ——, —— & Gomez Camba, J., 822 (Hel.)
di Matteo, J., with Claisse, Deuil & de Traverse, 970 (Leish.)
Mattingly, P. F., (761), (1048) (Ent.)
——, with Chow, (1151) (Ent.)
Matumoto, M., 36 (Pl.)
Mauri, A. C. & Hadler, W. A., (566) (Lep.)
Maurice, A., with Gaud & Faure, 609 (Mal.)
Maury, P. H., with Méret & Rollier, 636 (Am.)
——, with Rollier, Noury & Weisgerber, 1128 (Lep.)
Mauzé, J. & Languillon, J., 171 (Hel.)
May, J. M., 888 (Chl.)
Mayer, K. & Strenzke, K., 1052 (Ent.)
Mazhar, M., with Fawdry, 1096 (Leish.)
Mazzotti, L., 42 (R.F.)
——, with Colorado Iris, Treviño & Domínguez, 55 (Hel.)
Mechali, D., 444, 1072 (Mal.)
——, with Gaud, 713 (Mal.)
——, with —— & Delrieu, 433 (Mal.)
Méchin, R. & Si Hassen, A., 723 (Leish.)
Mehlman, B., with von Brand & Baernstein, 479 (Hel.)
Mehlman, B., with von Brand & Tobie, 530, 964 (Tryp.)
Mehta, J. N. (160) (Lep.)
Mehta, R. D., 732 (Chl.)
Meiklejohn, G., with Clark & Lennette, 626 (Typh.)
de Meillon, B., 203, (930) (Ent.)
—— & Hardy, F., 502 (Ent.)
——, with Lurie, 901 (Hel.)
Mein, R. M. & Rosado, P. N. S., 235 (Mal.)
Meira, J. A., Behmer, O. A. & Bloise, W., 1010 (Hel.)
——, Elejalde, G. & Pontes, J. F., 821 (Hel.)
——, with Ferreira, 406 (Misc. Dis.)
—— & Galvão, A. L. A., (384) (Hel.)
Meirelles, M. M., 271 (Hel.)
Meissner, E., 204 (Ent.)
Meister, H., 216 (Reports, etc.)
Meleney, F. L. & Johnson, B. A., (756) (Der.)
Melfi, J., 193 (Vms.)
de Mello, E. R., Modi, C. J., Walsh, M. P. & Patel, J. C., (65) (Def. Dis.)
de Mello, I. F., (566) (Lep.), 607 (Mal.)
de Mello, P. J., with Rachou, 524 (Mal.)
de Mello, V. H., 431 (Mal.)
——, with Schiavi, 857 (Mal.)
Melnitzky, I., with Kassel, (891) (Am.)
Mendes, E., 193 (Vms.)
Mendez de la Vega, J., with Scrimshaw & Guzman, 827 (Def. Dis.)
Mendez Lemaitre, A., 283 (Der.)
Mendheim, H., with Schmidt, 183 (Hel.)
——, with —— & Hannak, 577 (Hel.)
——, with —— & Schneid, 62 (Hel.)
Meneghello, J. & Niemeyer, H., 395 (Def. Dis.)
——, with ——, 395 (Def. Dis.)
——, —— & Espinosa, J., 395 (Def. Dis.)
Menk, W. & Mohr, W., 614 (Mal.)
Menon, M. A. U., (1151) (Ent.)
Menon, P. S., with Ramalingaswami, 192 (Haem.)
Menzies, G. C., with Eads & Ogden, (838) (Ent.)
Mercadal Peyri, J., 722 (Leish.)
Meredith, J. S. & Patton, G. Q., 1125 (Lep.)
Méret, I., with Rollier & Maury, 636 (Am.)
Merle, F., 673 (Ulc.)
de Mesquita, A. P., 926 (Misc. Dis.)
Messent, J. J., 613 (Mal.)
Messerschmitt, J., (919) (Haem.)
——, with Aubry, Boulard, Portier & Massonnat, (919) (Haem.)
Metcalf, R. L. & March, R. B., (298) (Ent.)
Meunier, R., 731 (Pl.)

Meyer, H., 22 (Mal.)
Meyer, K. F., 253 (Pl.)
——, with Quan & Chen, 254 (Pl.)
Meyer, L. M., Suarez, R. M., Martinez, E. & Suarez, R. M., Jr., with Benitez Gautier, C., 916 (Sp.)
Meyer, R., 245 *bis* (Typh.)
Meythaler, F. & Schaible, E., 1066 (Mal.)
Mickelsen, O., with Keys, Brožek, Henschel, Taylor, Simonson, Skinner & Wells, 767 (B.R.)
Mickler, E. E., Jr. & Diggs, L. W., (580) (Haem.)
Micks, D. W., 113 (Mal.)
——, with Rigdon & Breslin, 714 (Mal.)
Mietzsch, F., 764 (Reports, etc.)
Mikolajczyk, E., with Przesmycki & Wojciechowski, 244 (Typh.)
Mikulaszek, E. & Wojciechowski, E., 1099 (Typh.)
Milanes, F., with Spies, Garcia Lopez, Lopez Toca & Reboredo, 400 (Haem.)
——, with ——, ——, ——, —— & Stone, 399 (Haem.)
Millar, C. A., with Pesigan, Basaca-Sevilla, Pangilinan, Saniel, Garcia, Banzon, Beltran & Putong, 1017 (Hel.)
——, with ——, Putong & Garcia, 1015 (Hel.)
—— & Putong, P. B., 1018 (Hel.)
Miller, A., 1159 (B.R.)
Miller, J. K., 355 (Typh.)
Miller, L. A., (929) (Ent.)
Miller, M. J. & Gilani, A., 1109 (Am.)
Miller, T. B., with Aikawa & Harrell, 915 (Hel.)
Miloradovskaja, B. A., with Grennaus & Troitskij, 611 (Mal.)
Mingo Alsina, J. L., with de Prada & Gil Collado, 1101 (Typh.)
Mini, M., (822) (Hel.)
Ministry of Transport, 12 (Mal.)
Minnich, V., with Lange & Moore, 832 (Haem.)
Minning, W. & Ding, P., 748, 1035 (Hel.)
Minor, J. V., Jr., with Powell, Snyder, Benson & Woodward, 979 (Typh.)
Miranda, J. L., with Lins, Guimarães & Vasconcellos, 160 (Ys.)
Miranda, M., with Neghme, Agosin & Sanz, 738 (Am.)
de Miranda, S., 431 (Mal.)
Miró Carbonell, J., 815 (Lep.)
Mirri, A., 251 (Typh.)
Misra, S. S., 583 (Misc. Dis.)
Missiroli, A., 16 (Mal.)
Mitchison, J. M., with Perutz, (191) (Haem.)
Mitlin, N., with Gersdorff, (932), 1155 (Ent.)
Mitra, H. N., 927 (Misc. Dis.)
Miura, A., with Sasa, Kano, Hayashi, Kimura, Oyama, Sato, Hosoya & Hasegawa, 588 (Ent.)
Moçambique, 339 (Tryp.)
Modi, C. J., with de Mello, Walsh & Patel, (65) (Def. Dis.)
Modic, J. L., with Hunter, Dillahunt, Faust, Connellan, Bennett & Ingalls, 570 (Hel.)
Mofidi, C., with Ansari, 972 (Leish.)
——, with —— & Chafa, 969 (Leish.)
——, with —— & Chodja-Zade, 351 (Leish.)
——, with Baltazard & Bahmanyar, 367 (R.F.)
Moggridge, J. Y., 134 (Tryp.)
Mohan, B. N., (929) (Ent.)
Mohr, C. O., 883 (Typh.)
Mohr, W., with Menk, 614 (Mal.)
Mok, F. F., 874 (Mal.)
Mole, R. H., with Horne, 79, 923 (Heat Str.)
Molina S., M., with Campos R. de C., 371 (Lep.)
Möllhausen, W., 107 *bis* (Mal.)
Mollinedo, R., with Bolgert & Montel, 45 (Lep.)
Monnet, A., (134), 531 (Tryp.)
—— & Baylet, R., 621, 958 (Tryp.), 674 (Misc. Dis.)
Montalván C., J. A., 856, 867 (Mal.), 1091 (Tryp.)
——, with Jervis, 346 (Tryp.)
——, with Rodriguez M. & Gomez Lince, 382 (Hel.)
Montel, L. R., 472 (Ys.), 539 (Typh.), 741 (Lep., (1145) (Misc. Dis.)
——, with Bolgert & Mollinedo, 45 (Lep.)
——, Le Gac, P. & Mulet, M., 472, 739 (Ys.)
Montestruc, E., 880 (Typh.)
—— & Blaché, R., 270, 1127 (Lep.)
——, with Jullien-Vieroz & Caubet, 52 (Hel.)
Montézin, G., with Schneider, 265 (Am.)
Mooney, F. S., 1038 (Haem.)
Moore, C. V., with Lange & Minnich, 832 (Haem.)
Moore, D. E., with Burrows & Deupree, 258 *bis* (Chl.)
Moore, J. B., 211 (Ent.)
Mooser, H., 586 (Prot.)
Moreau, P., 140 (Typh.)
Morell Cuéllar, L. & González Castro, J., 1133 (Hel.)
Moretti, G. F., 120 (Mal.), 638 (Am.)
——, with Bonnin, 635 (Am.)
—— & Catros, P., 632 (Am.)
—— & Desnues, P., 638 (Am.)
Morgan, R. J., with Lamb, Shelmire, Cooper & Keaty, 71 (Der.)
Morin, H. G., 524 (Mal.)
Morishita, F., with Clark, 502 (Ent.)
Morlan, H. B., with Hill, Utterback & Schubert, 976 (Typh.)
Morris, M. G., 135 (Tryp.)
Morris, R. M., 298 (Reports, etc.)
Morrison, H. E., Lauderdale, R. W., Crowell, H. H. & Mote, D. C., (590) (Ent.)
Morrison, M. E., with Reese & Fowler, 142 (Bart.)
Mortenson, E. W., 929 (Ent.)
——, with Thurman, 677 (Ent.)
Morton, T. C., Neal, R. A. & Sage, M., 733 (Am.)
Moscoso Carrasco, C., (1063) (Mal.)
——, with Heriberto Tórres, (705) (Mal.)
Mosebach, E., 1154 (Ent.)
Moser, C. M., with Paulshock, (236) (Mal.)
Most, H., 520 (Mal.)
—— & van Assendelft, F., 991 *bis* (Am.)
——, with Tobie, Reardon & Bozicevich, 1110 (Am.)
Mote, D. C., with Morrison, Lauderdale & Crowell, (590) (Ent.)
Motta-Diana, A., with Buonomini & Maccarrone, 230 (Mal.)
Mouchet, R., 763 (Reports, etc.)
Mounter, L. A., (1038) (Vms.)
Mourant, A. E., with Donegani, Ibrahim & Ikin, (280) (Haem.)
Mozley, A., 1055 (B.R.)
Mozziconacci, P., with Debré, Bertrand & Bargeton, 1147 (Prot.)
Mücke, D., with Hudemann, 558, 812 (R.F.)

Mudrow-Reichenow, L., 871 (Mal.)
——, with Kikuth, 271 (Lep.)
Muench, H., with Barlow, 1005 (Hel.)
Mughairy, M. S., with Holmes, Gee, Asea, Bhima, Chagula, Evelia, Kasirye, Kiboneka, Lugazia, Majale, Ochieng, Gesa & Magambo, 397 (Haem.)
Mühlpfordt, H., with Westphal, 201 (Prot.)
Muir, E., 165, 375, 1127 (Lep.)
Muirhead-Thomson, R. C., 512, 704 (Mal.)
Mukerjee, N., with Dharmendra, 165 (Lep.)
Mukerji, A. K. & Mathen, K. K., 174 (Hel.)
Mukherji, A., with Ghosh, 152 (Am.)
Mukherji, S. P., with Seal, 985 (Pl.)
Mulet, M., with Le Gac & Lagarde, 496 (Der.)
——, with Montel & Le Gac, 472, 739 (Ys.)
Mulhern, T. D., with Peters, Thurman & Markos, 91 (Ent.)
Müller, G., 803 (Rab.), 1137 (Hel.)
——, with Vivoli, 988 (Am.)
Müller, W., with Heilmeyer & Schubothe, 831 (Haem.)
Mulligan, H. W., Sommerville, T. & Lloyd, O. C., 21 (Mal.)
Muñiz, A. I., with Asenjo, de Borinquen Segundo & Canals, (390) (Def. Dis.)
Muniz, J., 967 (Tryp.)
—— & dos Santos, M. C. F., 965, 966 (Tryp.)
Muñoz T., F., with Neghme, Bertín, Donckaster, Silva, Artigas, Bunster, Faúndes & Basso, 484 (Hel.)
Munsell, H. E., Williams, L. O., Guild, L. P., Troescher, C. B., Nightingale, G. & Harris, R. S., (751) (Def. Dis.)
Muraz, 844
Murgatroyd, F., 81 (Misc. Dis.), 177 (Hel.)
de Muro, P. & Leonardi, G., 752 (Haem.)
Murphy, J. B., with Batte & Swanson, 572 (Hel.)
Murray, E. S., with Pike & Cohen, 546 (Typh.)
——, with Sleisenger & Cohen, 728 (Typh.)
—— & Snyder, J. C., 536 (Typh.)
Muspratt, J., 1049 (Ent.)
Mustafa, D., 50 (Hel.)
Muth, J., with Schwarz, (497) (Der.)
Müting, L., with Gärtner, 486, 904 (Hel.)

N

Nagai, A., with Inoki, Kitamura, Takada & Nakabayashi, 633 (Am.)
Naik, R. G., Shah, M. T. & Nargund, K. S., (566) (Lep.)
Nair, C. P., 331, (950) (Mal.)
——, with Jaswant Singh, 1063 (Mal.)
——, with ——, Raghavan & Basu, 713 (Mal.)
——, with —— & Ray, 20, 334 (Mal.), 94 (Lab.)
Nájera, L. & Conejos, M., (745) (Hel.)
Nájera, L. E., 415, 1049 (Ent.)
Najib Khan, see Khan, N.
Nakabayashi, T., with Inoki, Nagai, Kitamura & Takada, 633 (Am.)
Nakagami, S., with Kondo, Yaoi & Nakano, 459 (Rab.)
——, with Nakano, Kondo & Yaoi, 459 (Rab.)
——, with Yaoi, Nakano & Kondo, 34, 459 (Rab.)
Nakamura, M. & Anderson, H. H., 1108 (Am.)
——, with Hrenoff, 992 (Am.)
Nakano, M., Kondo, A., Yaoi, H. & Nakagami, S., 459 (Rab.)
——, with ——, —— & ——, 459 (Rab.)
——, with Yaoi, Nakagami & Kondo, 34, 459 (Rab.)
Nakkash, B. Y., with Jalili & Taj-el-Din, 751 (Haem.)
Nardy, R. V., with Collins, 681 (Ent.)
Nargund, K. S., with Kshatriya & Patel, (523) (Mal.)
——, with Naik & Shah, (566) (Lep.)
Nature, 684 (Reports, etc.)
Nauck, E. G., 212 (Reports, etc.), 282 (Der.), 1057 (B.R.)
Navarranne, P., with Camain & Ayite, 819 (Hel.)
Navarro, L., with Downs & Bordas, 1157 (Ent.)
Navarro, R. J. & Santiago, N., 779 (Mal.)
de Navasquez, S., 81 (Misc. Dis.)
Neal, R. A., 264, 640 (Am.)
——, with Morton & Sage, 733 (Am.)
Neeb, H., 192 (Haem.)
Néel, R., 629 (Pl.)
Neghme, A., Agosin, M., Christen, R. & Rubio, M., 1114 (Am.)
——, Bertín, V., Donckaster, R., Silva, R., Artigas, J., Bunster, A., Muñoz T., F., Faúndes, A. & Basso, B., 484 (Hel.)
——, ——, Tagle, I., Silva, R. & Artigas, J., 484 (Hel.)
——, with Fanta & Faiguenbaum, (822), 822 (Hel.)
——, Miranda, M., Agosin, M. & Sanz, R., 738 (Am.)
—— & Silva, R., 155 (Am.)
Negroni, P., 1039 (Der.)
——, Vivoli, D. & Bonfiglioli, H., 1039 (Der.)
Neild, F. G., 443 (Mal.)
Nelson, N., with Eichna, Park, Horvath & Palmes, 403 (Heat Str.)
Nelson, R. H., with Fales, Fulton & Bodenstein, 1158 *bis* (Ent.)
Neto, J. A. F., with Rachou, 516 (Mal.)
Nettel F., R., 182 (Hel.)
Neujean, G., 956 (Tryp.)
Neva, F. A., with Kass & Finland, 725 (Typh.)
Newsome, J., 1006 (Hel.)
—— & Halawani, A., 48 (Hel.)
Newton, J. K., with Hentel & Horsman, 671 (Der.)
Nicholas, H. O., with Chandler & Read, (383) (Hel.)
Nicholls, L., 688 (B.R.)
Nicosia, J., with Herrera & Elton, 730, 801 (Y.F.)
Niemeyer, H. & Meneghello, J., 395 (Def. Dis.)
——, with ——, 395 (Def. Dis.)
——, with —— & Espinosa, 395 (Def. Dis.)
Nieves, M., with Ruiz Sanchez, Quezada, Paredes & Riebeling, 1069 (Mal.)
Nightingale, G., with Munsell, Williams, Guild, Troescher & Harris, (751) (Def. Dis.)
Nightingale, R. P., with Walt & Willis, 394 (Def. Dis.)
Ninard, B., 245 (Typh.)
Niño, F. L., 73 (Der.)
——, with Mackinnon, Artagaveytia-Allende, Vinelli, Ferrada-Urzúa, Alonso & Donoso, 582 (Der.)
——, Risolía, A. & Ferrada Urzúa, L., 74 (Der.)
Noël, R. & Marie-Suzanne, 652 (Lep.)

Nogueira, J. F. P. & Coito, A. de M. F., 384 (Hel.)
Nogueira, J. P. & Coito, A. M., 419 (Reports, etc.)
——, Coito, A. de M. & Ferreira, A. E., 280 (Haem.)
Nolan, M. O., 273 (Hel.)
——, with Berry & Oliver González, 53 (Hel.)
van Noort, H. R., 843 (B.R.)
Nor El-Din, G., 637 (Am.)
Norsen, J., with Felsenfeld, Soman, Ishihara & Waters, 988 (Chl.)
Norton, L. B., with Pimentel & Schwardt, 413, 680 (Ent.)
Nour el-Din, G., (387) (Hel.)
Noury, with Rollier, Weisgerber & Maury, 1128 (Lep.)
Noyan, A., 606, 861, 1069 (Mal.)
Nuñez Andrade, R., 753 (Der.)
Nwokolo, C., 60 (Hel.)

O

Obrador Alcalde, S., with Alés Reinlein & Arjona Trigueros, 1133 (Hel.)
O'Brien, J. P., 925 (Heat Str.)
Ochieng, E. C., with Holmes, Gee, Asea, Bhima, Chagula, Evelia, Kasirye, Kiboneka, Lugazia, Majale, Mughairy, Gesa & Magambo, 397 (Haem.)
O'Connor, G. R., (747) (Hel.)
Oehme, J., 42 (R.F.)
Oelkers, H. A., 489 (Hel.)
Offutt, E. P., with Hahn, 175 (Hel.)
Ogden, L. J., with Eads & Menzies, (838) (Ent.)
O'Hara, H., with Goldman, 282 (Der.)
Olarte, J., with Watson & Angulo, 194 (Der.)
Oliveira, J., with Fonseca, Pinto, Colaço, Branco, da Gama, Franco & Lacerda, 980 (Typh.)
Oliver González, J., with Berry & Nolan, 53 (Hel.)
——, Biaggi, N., Acevedo, C., Mariotta, D. A., Hernández Morales, F., Santiago Stevenson, D. & Pérez Santiago, E., 54 (Hel.)
—— & Thillet, C. J., 53 (Hel.)
Oliveros, S. B., with Salcedo, Bamba, Carrasco, Chan, Concepcion, Jose, de Leon, Pascual, Santiago & Valenzuela, 494 (Def. Dis.)
Olivier, L. J. G., 326 (Mal.)
bin Omar, A. H., with Reid, 332 (Mal.)
Onabamiro, S. D., 1032 (Hel.)
Onetto S., R., with Velasco & Escobar, (246) (Typh.)
Oomen, H. A. P. C., 829 (Def. Dis.)
Ordman, D., 922 (Vms.)
Orenstein, A. J., (58) (Hel.)
Ormerod, W. E., 961, 962 (Tryp.)
O'Rourke, F. J., with Crewe, 907 (Hel.)
Orser, W. B. & Brown, A. W. A., (594) (Ent.)
Ortiz Mariotte, C., with Varela, (795) (Typh.)
Osbond, J. M., with Fulton, Johner, King & Wright, 365 (Am.)
Osimani, J. J., 1156 (Ent.)
Oswald, N., 583 (Misc. Dis.)
Otto, G. F., with Jachowski & Wharton, 824 (Hel.)
Ovanessian, G., with el Zahawi, 58 (Hel.)
Ovazza, M., with Grenier, 761 (Ent.)
Overman, R. R., Hill, T. S. & Wong, Y. T., 118 (Mal.)
Oyama, K., with Sasa, Kano, Hayashi, Kimura, Miura, Sato, Kosoya & Hasegawa, 588 (Ent.)
van Oye, E. & Peel, E., 792 (Tryp.)

P

Paez, H., 1031 (Hel.)
Pagan, C. & Hageman, R. H., 206 (Ent.)
Pagast, F., with Eichler, 15 (Mal.)
——, with —— & Löpmann, 1064 (Mal.)
Pagès, R. & Gaud, J., (1137) (Hel.)
Paillas, J. E. & Tamalet, J., (745) (Hel.)
Pal, R., 590 (Ent.)
Palmes, E. D., with Eichna, Park, Nelson & Horvath, 403 (Heat Str.)
Pampana, E. J., 782 (Mal.)
Pampiglione, S., (401) (Der.)
Pan, C. & Hunter, G. W., 1012 (Hel.)
——, Kaufman, E. H. & Hunter, G. W., 1019 (Hel.)
Pandit, C. G. & Hora, S. L., 1106 (Chl.)
Pandit, S. R., 457 (Rab.)
Pangilinan, M. V., 1018 (Hel.)
——, with Pesigan, Basaca-Sevilla, Saniel, Garcia, Banzon, Beltran, Putong & Millar, 1017 (Hel.)
——, with ——, Saniel, Garcia, Banzon & Putong, 1018 (Hel.)
——, with —— & Sarmiento, 656 (Hel.)
Panja, D., Gupta, J. C. & Banerji, A. K., 581 (Der.)
——, with Sen Gupta & Banerjee, 454 (Leish.)
Pansa, G., with Benetzzo, (476) (Hel.)
Pará, M., 857 (Mal.)
Paracampos, H., with de Bustamante, Lacerda & Santos, 319 (Mal.)
Paraense, W. L. & Santos, J. M., 272, 478 (Hel.)
Paras, E. M., 645 (Lep.)
Pardo-Castello, V., Trespalacios, F., Tiant, F. R., Ibarra, R., Jose Mestre, J. & Beade, P., 652 (Lep.)
Paredes, M., with Ruiz Sanchez, Nieves, Quezada & Riebeling, 1069 (Mal.)
Parent, M., with Lenelle, 1138 (Def. Dis.)
Parenzan, P. & Chieffi, G., 1051 (Ent.)
Paris, P., with Sohier & Camain, (1023) (Hel.)
Park, C. R., with Eichna, Nelson, Horvath & Palmes, 403 (Heat Str.)
Parker, A. H., (92) (Ent.)
Parker, R. R., Pickens, E. G., Lackman, D. B., Bell, E. J. & Thraikill, F. B., 796 (Typh.)
Parker, R. T., with Bauer, Hall, Benson, Joslin, Hightower, Snyder, Venable & Woodward, 979 (Typh.)
——, Bauer, R. E., Lister, L. M., Woodward, T. E. & Hall, H. E., 540 (Typh.)
Parrot, L., 761, (930) (Ent.)
——, with Sergent, 516 (Mal.)
Pascale, H., 540 (Typh.), 817 (Hel.)
Pascual, C. R., with Salcedo, Bamba, Carrasco, Chan, Concepcion, Jose, de Leon, Oliveros, Santiago & Valenzuela, 494 (Def. Dis.)
Passalacqua, C. de Sá P., (431) (Mal.)
Patel, J. C., with de Mello, Modi & Walsh, (65) (Def. Dis.)
—— & Varde, A. P., 580 (Haem.)
Patel, S. R., with Kshatriya & Nargund, (523) (Mal.)
Patterson, P. M., with Zumpt, (502) (Ent.)
Patton, G. Q., with Meredith, 1125 (Lep.)
Paul, J. H., Watson, R. B. & Liang, K. C., 443 (Mal.)
de Paula, A., 637 (Am.)
Paulley, J. W., with Hart, Rivers & Westlake, 1145 (Prot.)

Paulshock, M. & Moser, C. M., (236) (Mal.)
Pautrizel, R., Rivasseau, J. & Rivasseau, D., 1133 (Hel.)
Pavilanis, V., with Lépine & Atanasiu, 887 (Rab.)
Payet, M., 430 (Mal.)
Payne, E. H., Bellerive, A. & Jean, L., 1118 (Ys.)
——, with Pijoan & Dineen, 758 (Oph.)
——, with —— & Loe, 1043 (Oph.)
Payzin, S., 30 (Typh.)
Pearson, F. A., 763 (Reports, etc.)
Peel, E., with van Oye, 792 (Tryp.)
Peer, K. C., with deOng & Fancher, 297 (Ent.)
Pellegrino, J., 450 (Tryp.)
Penati, F. & Randone, G., 543 (Typh.)
Penido, H. M., de Sousa, E. F. & Bezerra, F. P. G., Jr., 235 (Mal.)
Pennec, J., 165 (Lep.)
Penner, L. R., 172 (Hel.)
Pereira, H. G., Cardoso, H. & Travassos, J., 979 (Typh.)
Pérez, R., with Beltrán, 87 (Prot.)
Pérez Santiago, E., with Oliver González, Biaggi, Acevedo, Mariotta, Hernánez Morales & Santiago Stevenson, 54 (Hel.)
Perlowagora, A., with Hughes, 547 (Y.F.)
Perrin, M., 240, (532) (Tryp.)
Perry, W. J., 112 (Mal.)
Perutz, M. F., Liquori, A. M. & Eirich, F., (1142) (Haem.)
Perutz, M. F. & Mitchison, J. M., (191) (Haem.)
Pesigan, T. P., 274, 1014, 1019 (Hel.)
—— & Banzon, T. C., 1016 (Hel.)
——, Basaca-Sevilla, V., Pangilinan, M. V., Saniel, V. F., Garcia, E. G., Banzon, T., Beltran, A., Putong, P. B. & Millar, C. A., 1017 (Hel.)
—— & Beltran, A. M., 1016 (Hel.)
——, Pangilinan, M. V., Saniel, V. F., Garcia, E. G., Banzon, T. C. & Putong, P. B., 1018 (Hel.)
——, —— & Sarmiento, A. P., 656 (Hel.)
——, Putong, P. B., Garcia, E. G. & Millar, C. A., 1015 (Hel.)
Peterkin, G. A. G., with Conrad & Manson, 623 (Leish.)
Peters, D., with Wigand, 141 (Bart.)
Peters, L., with Bueding, 654 (Hel.)
—— & Wright, H. N., 964 (Tryp.)
Peters, R. F., with Gray & Washburn, 292 (Ent.)
——, Thurman, D. C., Jr., Markos, B. G. & Mulhern, T. D., 91 (Ent.)
Petit, with Rollier & Rollier, (995) (Lep.)
Petrilli, F. L., 56, 57 (Hel.)
Petrow, V. & Wragg, W. R., (342) (Tryp.)
Pétrus, E. A., with Rein & Kitchen, 158 (Ys.)
Pettazzi, A., 41 (Am.)
Philen, E. A., with Krusé & Ludvik, 615 (Mal.)
Phillips, B. P., 1107 (Am.)
Philpot, V. B. & Smith, R. G., 69 (Vms.)
Phipps, J., 471 (R.F.)
Phipps, P. H., with Marmion, MacCallum, Rowlands, Thiel & McDonald, 1100 (Typh.)
Piaggio Blanco, R. A., Dubourdieu, J. J., Dighiero, J., Joaquin Canabal, E. & Grosso, O. F., (56) (Hel.)
Picaso Guillen, J., 108 (Mal.)
Pick, F., 90 (Ent.), 172, 484, (574), 659 *bis* (Hel.), 448 (Mal.)
Pickens, E. G., with Parker, Lackman, Bell & Thraikill, 796 (Typh.)
Piekarski, G., 586, 675 (Prot.)
—— & v. Törne, H., 500 (Prot.)
Pieraerts, G., 1036 (Def. Dis.)
Pierquin, L., 558 (R.F.)
——, with Himpe, 125 (Mal.), 1116 (R.F.)
Pietri, H., with Thiodet, Fourrier, Massonat & Aubry, 992 (Am.)
di Pietro, P., 262 (Am.), 272 (Hel.)
Piette, M., with Siguier, Choubrac & Herman, 155, 468 (Am.)
——, with ——, —— & Sebaoun, 808 (Am.)
——, with —— & Crosnier, 38 (Am.)
Piganiol, Fus & Rajaobelina, 926 (Ulc.)
Pijoan, M., Loe, F. & Payne, E. H., 1043 (Oph.)
——, Payne E. H. & Dineen, J., 758 (Oph.)
Pike, G., Cohen, S. & Murray, E. S., 546 (Typh.)
Pillat, A., (1150) (Prot.)
Pille, G., 621 (Tryp.)
Pimentel, D., Schwardt, H. H. & Norton, L. B., 413, 680 (Ent.)
Pinero Carrillo, J. F., with Choza Ferrer, 106 (Mal.)
Pinkerton, H., with Laskowski & Stanton, 730 (Bart.)
——, with Stanton & Laskowski, 143 (Bart.)
Pinotti, M., 1082 (Mal.)
Pinto, A. R., with Ferreira & de Almeida, 341 (Tryp.)
——, with Trincão, de Almeida & Gouveia, 281 (Haem.)
Pinto, J. T. & Zeo, A., 1121 (Lep.)
Pinto, M. R., with Fonseca, Colaço, Oliveira, Branco, da Gama, Franco & Lacerda, 980 (Typh.)
Pipkin, C. A., with Berberian & Dennis, 154 (Am.)
Pirastu, C., with Deriu, 148 (Am.), 466 (Am.)
Pires, F. A., 955 (Tryp.)
——, da Silva, J. M. & Teles e Cunha, G., 448 (Tryp.)
Pires, F. M., with Ferreira & de Almeida, 341 (Tryp.)
Pires, W. da M., with de Bustamante, 501 (Ent.)
——, with —— & Gusmão, 617 (Mal.)
Piringer, W. A., 145 (Rab.)
Pittaluga, J. D., with Blanco & Resta, 364 (Am.)
Pittendrigh, C. S., 115 (Mal.)
Pizzi, T., with Jarpa, Christen & Agosin, 1096 (Tryp.)
Pizzi P., T., 531 (Tryp.)
Pizzillo, G., 234, 325, 518, 709, 864 (Mal.), 455 (Leish.), 1102 (Typh.)
Plackett, R. L., with Hewlett, (212) (Ent.)
Plagnol, H., 683 (Ent.)
Plooij, M. & Wüstefeld, P., (552) (Am.)
Pöhn, H. P., with Brandis, 552 (Am.)
Poindexter, H. A., 369 (Lep.)
Poirier, M., with Deschiens, 571, 658 (Hel.)
Pokorny, M. & Klausová, L., 177 (Hel.)
Poleff, L., 672 (Oph.)
Pollitzer, W. A., with Redmond & Runyon, 715 (Mal.)
Pollock, G. A., with Kirwan, Langrishe & Martin, 939 (B.R.)
Polson, A., (1091) (Tryp.)
Polunin, I., 1029 (Hel.)
Poncet, A., with Sergent, 445 (Mal.)

Pons, E. R., Jr., with Diaz Rivera & Ramirez, 404 (Misc. Dis.)
Del Ponte, E., with Alberto Alvarado, 115 (Mal.)
Pontes, J. F., with Meira & Elejalde, 821 (Hel.)
Le Poole, A., 1009 *bis* (Hel.)
Popli, S. P., with Clemo, (864) (Mal.)
Porter, J. E. & Hughes, J. H., (205) (Ent.)
Portères, R., (579) (Def. Dis.)
Portier, A., with Aubry, Boulard, Messerschmitt & Massonnat, (919) (Haem.)
Portugal, H. & Azulay, R. D., 563 (Lep.)
Poti, S. J., 1070 (Mal.)
Potter, C., with Lord, (933) (Ent.)
Potts, W. H., 951 (Tryp.)
Poul, J., (455) (Leish.)
——, with Donatien & Gayot, (804) (Rab.)
Powell, A. M., Snyder, M. J., Minor, J. V., Jr., Benson, J. F. & Woodward, T. E., 979 (Typh.)
Powell, R. G., with Haworth, McKenna & Woodward, (894) (Am.)
de Prada, J., Gil Collado, J. & Mingo Alsina, J. L., 1101 (Typh.)
Do Prado, F. C., 554 (Am.)
Prado, R., with Cariola, Agosín & Christen, 451 (Tryp.)
Prakash, S., with Ramakrishnan, 618 (Mal.)
Prasad, B. G., 915 (Def. Dis.)
Prather, R. M., Jr., with Becker & Schwink, 1085 (Mal.)
Pratt, J. J., Jr. & Babers, F. H., 207 (Ent.)
Previtera, G. & Condorelli, V., (27) (Typh.)
Price, I. N. O., with Fulton & Joyner, 734 (Am.)
Prieto Lorenzo, A., with Matilla & Aparicio Garrido, 573, 662 (Hel.)
——, with ——, —— & Diez Melchor, 176 (Hel.)
——, with ——, —— & Gomez Camba, 822 (Hel.)
Pringle, G., with Watson, 480 (Hel.)
——, with —— & Jamil, 1005 (Hel.)
Proença, L. M., with Schiavi, 333 (Mal.)
Proto, P., 363 (Am.)
Provost, A., with Roubaud, 1087 (Tryp.)
Przesmycki, F., Wojciechowski, E. & Mikolajczyk, E., 244 (Typh.)
Puchol, J. R., with Gomez Orbaneja, 998 (Lep.)
Purandare, N. M., with Bhende, Banker, Figueredo & Desai, 452 (Leish.)
Purvis, J. T., 598 (Reports, etc.)
Putkonen, T., with Telkkä, Teir & Hortling, 740 (Lep.)
Putong, P. B., with Millar, 1018 (Hel.)
——, with Pesigan, Basaca-Sevilla, Pangilinan, Saniel, Garcia, Banzon, Beltran & Millar, 1017 (Hel.)
——, with ——, Garcia & Millar, 1015 (Hel.)
——, with ——, Pangilinan, Saniel, Garcia & Banzon, 1018 (Hel.)
Puyelo, M. R., (908) (Hel.)
Puyuelo, R., 182, 278 (Hel.)

Q

Qazilbash, N. A., (567) (Hel.)
Quan, S. F., Chen, T. H. & Meyer, K. F., 254 (Pl.)
Quattlebaum, J. E., with Rigdon, 620 (Bl.)
Quesnel, J. J., with Buck, Courdurier & Dorel, (1097) (Leish.)
Quevauviller, A., (325) (Mal.)
Quezada, M., with Ruiz Sanchez, Nieves, Paredes & Riebeling, 1069 (Mal.)
Quinby, G. E., 512 (Mal.)
——, with Andrews & Langmuir, 430 (Mal.)
Quinland, W. S. & Chenault, J. W., (72) (Der.)
Quraishi, M. S., Ahmed, M. & Gramiccia, G., 1064 (Mal.)

R

Rabenko, J., with de Soldati, 878 (Tryp.)
Rabenschlag, K., with Laur, 729 (Typh.)
Rachou, R. G., 1066 (Mal.)
——, Ferreira, M. O. & Lima, M. M., 618 (Mal.)
—— & Gaberlini, J., Jr., 434 (Mal.)
—— & de Mello, P. J., 524 (Mal.)
—— & Neto, J. A. F., 516 (Mal.)
——, Ricciardi, I. & Guedes, A. S., 324 (Mal.)
—— & de Sousa, J., 411 (Ent.)
Radke, R. A., 808, 891, 893 (Am.)
Radkowiak, J. D., 724 (Typh.)
Raffaele, G. & Baldi, A., 788 (Mal.)
—— with Coluzzi, 1154 (Ent.)
Rafi, S. M. & Shah, I, A., 1071 (Mal.)
Rageau, J., 1088 (Tryp.)
Raghavan, N. G. S., 137 (Leish.)
—— with Jaswant Singh, Nair & Basu, 713 (Mal.)
—— & Satya Prakash, 137 (Leish.)
Rai Chaudhuri, M. N., with Chaudhuri & Ghosal, 255 (Chl.)
——, with ——, Ghosh & Dutta, 711 (Mal.)
Rajaobelina, with Piganiol & Fus, 926 (Ulc.)
Rajendram, S., Cader, M. H. M. A. & Visvalingham, T., 17 (Mal.)
Ramage, J. S., 381 (Hel.)
Ramakrishnan, S. P., with Jaswant Singh & David, 878 (Tryp.)
—— & Prakash, S., 618 (Mal.)
Ramalingaswami, V. & Menon, P. S., 192 (Haem.)
—— & Ventkatachalam, P. S., 190 (Haem.)
Ramamurthi, B., with Raman & David, 55 (Hel.)
Raman, T. K., Ramamurthi, B. & David, C. V., 55 (Hel.)
Ramanujam, K. & Smith, M., 374 (Lep.)
Ramirez, E., with Diaz Rivera & Pons, 404 (Misc. Dis.)
Ramirez-Rodriguez, E., with Rodriguez-Molina, Acevedo, Torres & Lopez-Sanabria, 481 (Hel.)
Ramsay, J. A., (677) (Ent.)
Randone, G., with Penati, 543 (Typh.)
Ranier, A., 836 (Der.)
Ranking, D. L., 250 (Typh.)
Ranque, J., with Sautet, Vague & Vuillet, 147 (Am.)
Ransom, S. E. & Huebner, R. J., 545 (Typh.)
Ranta, L. E. & McLeod, M., 358 (Chl.)
Rao, K. N. A., with Kalra & Jacob, 811 (R.F.)
Rao, T. R., 1075 (Mal.)
——, with Viswanathan, 127 (Mal.)
Rao, V. V., 126 (Mal.)
Raper, A. B., 1010 (Hel.)
—— & Ladkin, R. G., 196 (Misc. Dis.)
Rappaport, E. M., Rossien, A. X. & Rosenblum, L. A., 892 (Am.)
Rappaport, I. & Wells, H. S., 1034 (Hel.)
Ratcliffe, H. L. & Worth, C. B., 1048 (Prot.)
Rath de Souza, P. & de Souza Lima, M., 45 (Lep.)
Raoult, A., 668 (Def. Dis.)
Rausch, R. & Schiller, E. L., 661 (Hel.)
Rawson, G. W., with Bradner, 990 (Am.)

Ray, A. P., with Jaswant Singh & Nair, 20, 334 (Mal.), 94 (Lab.)
——, with Josephson, Taylor & Greenberg, 862 (Mal.)
——, with Taylor, Greenberg & Josephson, 873 (Mal.)
Raynal, J. H., 951 (Tryp.)
—— & Lotte, A. J., 960 (Tryp.)
Rayroux, J., with Schneider, 258 (Lep.)
Razemon, P., with Coutelen & Biguet, (275) (Hel.)
Read, C. P., (745), (821) (Hel.)
——, with Chandler & Nicholas, (383) (Hel.)
Reagan, R. L. & Brueckner, A. L., 457 (Rab.)
Reardon, L. V., with Rees, Bartgis, & Hellerman, 259 (Am.)
——, with Tobie, Most & Bozicevich, 1110 (Am.)
Reboredo, A., with Spies, Garcia Lopez, Milanes & Lopez Toca, 400 (Haem.)
—— with ——, ——, ——, —— & Stone, 399 (Haem.)
Reczko, E., 544 (Typh.)
Reddy, D. G. & Varmah, K., 660 (Hel.)
Redmond, W. B., Runyon, L. C. & Pollitzer, W. S., 715 (Mal.)
Reebstein, H., 610 (Mal.)
Rees, C. W., Reardon, L. V. & Bartgis, I. L., with Hellerman, L., 259 (Am.)
Reesal, M. R., (746) *bis* (Hel.)
—— with Fairbairn, 663 (Hel.)
Reese, J. D., Morrison, M. E. & Fowler, E. M., 142 (Bart.)
Reeves, W. C., 1151 (Ent.)
Rehm Schmidt, F., 351 (Leish.)
Rehn, J. W. H., Henderson, J. M. & Serrano, J. M., 113 (Mal.)
——, Maldonada Capriles, J. & Henderson, J. M., 113 (Mal.)
Reid, J. A., 867 (Mal.)
—— & Hodgkin, E. P., 321 (Mal.)
——, with bin Omar, A. H., 332 (Mal.)
Reid, J. D., with Blakeley, 661 (Hel.)
Rein, C. R., Kitchen, D. K. & Pétrus, E. A., 158 (Ys.)
Reinertson, J. W. & Thompson, P. E., 1113 (Am.)
Reingold, I. M., 402 (Der.)
Reisel, J. H. & Groen, J., 825 (Hel.)
Reiss, F., 1122 (Lep.)
Reitano, U. & Fondacaro, A., 573 (Hel.)
Remlinger, P. & Bailly, J., 1104 (Rab.)
Rempel, J. G., with Arnason, Brown, Fredeen & Hopewell, (92) (Ent.)
Resta, C. A., with Blanco & Pittaluga, 364 (Am.)
Reuter, S., with Ascher & Levinson, 1155 (Ent.)
Rhamy, R. K., with Robinson & Kincaid, 196 (Heat Str.)
Rhodesia, Southern, 298 (Reports, etc.)
Ribas, E. B. & Tavares, A., 560 (Ys.)
Ricci, M., with Alicata, 1033 (Hel.)
Ricciardi, I., with Rachou & Guedes, 324 (Mal.)
Richards, W. R., with Hocking & Twinn, (90) (Ent.)
Richeson, E. M., with Greenberg, 950 (Mal.)
Rickard, E. R., 726 *bis*, 796, 882 (Typh.)
—— & Worth, C. B., 976 (Typh.)
——, with ——, 881, 882 (Typh.)
Rico-Avello y Rico, C., 854 (Mal.)
Ricossé, J., with Crosnier, Darbon, Ducourneau, Bouilloc & Martin, 991 (Am.)
Riddell, W. A., with Taylor, 377 (Hel.)
Ridi, U., Signorini, L. F., Davoli, R. & Ceccherini, M., 799 (Typh.)
Riebeling, R., with Ruiz Sanchez & Arreola, 39 (Am.)
——, with ——, Nieves, Quezada & Paredes 1069 (Mal.)
Riedel, B. B., 388 (Hel.)
Rieger, H., 928, (1146) (Prot.)
van Riel, J., 618 (Mal.)
Riemschneider, R., 1052 *bis* (Ent.)
—— & Rohrmann, B., 295 (Ent.)
Rigdon, R. H., 119 (Mal.)
—— & Breslin, D., 234 (Mal.)
——, Micks, D. W. & Breslin, D., 714 (Mal.)
—— & Quattlebaum, J. E., 620 (Bl.)
Riley, I. D. & Arneil, G. C., 407 (Prot.)
Rinaudo, E., with Roman, 683 (Ent.)
Rio Leon, E., 814 (Lep.)
Ríos, T., with Avilés Nugué, (1095) (Tryp.)
——, with Rodriquez M. & Villamizar, 387 (Hel.)
Risolía, A., with Niño & Ferrada Urzúa, 74 (Der.)
Rist, N., with Floch, 375 (Lep.)
Rivasseau, D., with Pautrizel & Rivasseau, 1133 (Hel.)
Rivasseau, J., with Pautrizel & Rivasseau, 1133 (Hel.)
Rivers, J. S., with Hart, Paulley & Westlake, 1145 (Prot.)
Roberts, E. W., 54 (Hel.)
Roberts, F. H. S., 515 (Mal.)
Robertson, K., 1090 (Tryp.)
Robertson, R. S., Jr. & Gaines, J. C., (932) (Ent.)
Robineau, A., 922 (Oph.)
Robinson, G. G., 143 (Y.F.)
Robinson, S., Gerking, S. D., Turrell, E. S. & Kincaid, R. K., 78 (Heat Str.)
——, Kincaid, R. K. & Rhamy, R. K., 196 (Heat Str.)
Rocha, A., with de Magalhaes, 541 (Typh.)
——, with —— & Café, 728 (Typh.)
—— with —— & Freire, (541) (Typh.)
Rocha, H. S., with Vieira, 1136 (Hel.)
Rocha e Silva, M., (71) (Vms.)
Rockefeller Foundation, 95 (Reports, etc.)
Rockenschaub, A., 184 (Hel.)
Rodaniche, A., with de Rodaniche, 28 (Typh.)
de Rodaniche, E. C. & Rodaniche, A., 28 (Typh.)
Rodhain, J., 870 (Mal.), (878) (Tryp.)
——, with de Coster, 1050 (Ent.)
—— & Vincke, I., 871 (Mal.)
Rodriguez, J. N., 644, 649 (Lep.)
——, with Uyguanco & Concepcion, 997 (Lep.)
Rodriguez M., J. D., 1093 (Tryp.)
——, Gomez Lince, L. F. & Montalvan, C., J. A., 382 (Hel.)
——, Villamizar, L. & Rios, T., 387 (Hel.)
Rodriguez-Molina, R., Acevedo, C. E., Torres, J. M., Lopez-Sanabria, U. & Ramirez-Rodriguez, E., 481 (Hel.)
Rodríguez Pavón, A., 567 (Hel.)
Rodríguez Velandia, O., with Bonilla Naar, 1107 (Am.)
Rohrmann, B., with Riemschneider, 295 (Ent.)
Rohwer, S. A. & Haller, H. L., (594) (Ent.)
Rojas, E., with Gajardo Tobar, Apablaza, Uribe, Benavides, Vargas, Cepeda, Zeldis, Lucchini & De la Fuente, 381 (Hel.)
Rollier, M., with Rollier & Petit, (995) (Lep.)

Rollier, R., Maury, P. H. & Méret, I., 636 (Am.)
——, Noury, Weisgerber & Maury, 1128 (Lep.)
——, Rollier, M. & Petit, (995) (Lep.)
Rollo, I. M., 1086 (Mal.)
——, with Falco, Goodwin, Hitchings & Russell, 872 (Mal.)
—— & Williamson, J., 955 (Tryp.)
Roman, E. & Rinaudo, E., 683 (Ent.)
Romeo, V., 230 (Mal.)
Romeo de León, J., with Antomio Cabrera, 349 (Leish.)
Romeo Viamonte, J. M., 778 (Mal.)
Romer, J. D., 549 (Pl.)
Romero, L., A., 1000, 1119, 1129 (Lep.)
——, Castro Jenkins, A. & Alvarado, R., 1123 (Lep.)
Rosa, B., with d'Ignazio, 35 (Rab.)
Rosado, P. N. S., with Mein, 235 (Mal.)
Del Rosario, F. G., with Smith & Dy, 112 (Mal.)
Rose, F. L., with Crowther & Curd, (864) (Mal.)
——, with ——, ——, Davey, Hendry & Hepworth, (864) (Mal.)
Rose, H. M., 978 (Typh.)
——, Kneeland, Y., Jr. & Gibson, C. D., 547 (Typh.)
Rosemberg, J., Aun, J. N. & de Souza Campos, N., 373 (Lep.)
——, Souza Campos, N. & Aun, J. N., 1121 *bis* (Lep.)
——, with —— & ——, 372 (Lep.)
Rosen, E. & Belber, J. P., 671 (Der.)
Rosenblum, L. A., with Rappaport & Rossien, 892 (Am.)
Rosenstiel, with Hillemand, 153 (Am.)
Ross, H., 376, 997 (Lep.)
Ross Institute Industrial Advisory Committee, 568 (Hel.)
Ross, R. W., 802 (Y.F.)
—— & Gillett, J. D., 627 (Y.F.)
——, with ——, Dick, Haddow & Hewitt, 627 (Y.F.)
Ross, S. G., 393 (Def. Dis.)
Rossien, A. X., with Rappaport & Rosenblum, 892 (Am.)
Rotberg, A., Bechelli, L. M. & Keil, H., 266 (Lep.)
Roth, A. R., with Lindquist, Yates, Hoffman & Butts, 931 (Ent.)
Roth, H., 1034 (Hel.)
de Rothschild, B. & Lévy, G., 709 (Mal.)
Roubaud, E. & Bourdié, M., 344 (Tryp.)
—— & Provost, A., 1087 (Tryp.)
Roubert, L., with Horrenberger, 353 (Typh.)
Rouby, M., with Le Gac, 540 (Typh.)
Roux, H., with Lamy, 265 (Am.)
Row, R., (242) (Leish.)
Rowlands, A., with Marmion, MacCallum, Thiel, McDonald & Phipps, 1100 (Typh.)
Roy, A. R., with Tribedi, (990) (Am.)
Royer, J., with Farinaud & Choumara, 6 (Mal.)
Royer, P., (383) (Hel.)
Rozeboom, L. E., 93 (Ent.)
Różniecka, D., 1100 (Typh.)
Rubio, M., with Agosin & Christen, 156, 471 (Am.)
——, with Neghme, Agosin & Christen, 1114 (Am.)
Ruchman, I. & Fowler, J. C., 1150 (Prot.)
Rudat, K. D., 572 (Hel.)
Rudra, M. N. & Kant, L., 198 (Misc. Dis.)
Ruge, H., 125 (Mal.)
Ruiz Martínez, C., (1054) (Ent.)
Ruiz Sanchez, F., Nieves, M., Quezada, M., Paredes, M. & Riebeling, R., 1069 (Mal.)
——, Riebeling, R. & Arreola, C., 39 (Am.)
Runyon, L. C., with Redmond & Pollitzer, 715 (Mal.)
Russell, P. B., with Falco, Goodwin, Hitchings & Rollo, 872 (Mal.)
——, with —— & Hitchings, (949) (Mal.)
—— & Hitchings, G. H., (949) (Mal.)
Russell, P. F., 606 (Mal.)
Rutkovskij, L. A., 664 (Hel.)
Ryrie, G. A., 648 (Lep.)

S

Sabatini, A., 1071 (Mal.)
Sabido, F., 453 (Leish.)
Sabin, A. B., 87 (Prot.)
—— & Feldman, H. A., 202 (Prot.)
Saccà, G., 839 (Ent.)
Sacktor, B., 591 (Ent.)
Sadun, E. H., Ané, J. N., Fuller, F. W. & Lewis, R., 261 (Am.)
——, Bradin, J. L., Jr. & Faust, E. C., 1112 (Am.)
——, with Faust, Everritt, Bradin & Lewis, 148 (Am.)
Safavi, G., with Baltazard & Bahmanyar, 157 (R.F.)
Sage, M., with Norton & Neal, 733 (Am.)
Saggese, E., (247) (Typh.)
Saito, M., 362 (Am.)
Saito, M. T., with Smith, Beard, Kepp, Clark & Eddie, 73 (Der.)
Saladino, E., 351 (Leish.)
Salcedo, J., Jr., Bamba, M. D., Carrasco, E. O., Chan, G. S., Concepcion, I., Jose, F. R., de Leon, J. F., Oliverso, S. B., Pascual, C. R., Santiago, L. C. & Valenzuela, R. C., 494 (Def. Dis.)
——, with Burch, Carrasco, Intengan & Caldwell, 64 (Def. Dis.)
Salis, H. & Smith, W. C., 570 (Hel.)
Salmon, T. N., with Starr & Ferguson, 211 (Ent.)
Sánchez Castellanos, M., 185 (Hel.)
Sandalio Miguel, D., 815 (Lep.)
Sandars, D. F., 747 (Hel.)
de Sande, A. G., 787 (Mal.)
Sandosham, A. A., 387, (907) (Hel.)
Sandstead, H. R., 766 (B.R.)
Saner, R. G. & Fehler, B. M., 356 (Typh.)
Sani, A., 318 (Mal.)
Saniel, V. F., 1018 (Hel.)
——, with Pesigan, Basaca-Sevilla, Pangilinan, Garcia, Banzon, Beltran, Putong & Millar, 1017 (Hel.)
——, with ——, Pangilinan, Garcia, Banzon & Putong, 1018 (Hel.)
Santiago, L. C., with Salcedo, Bamba, Carrasco, Chan, Concepcion, Jose, deLeon, Oliveros, Pascual & Valenzuela, 494 (Def. Dis.)
Santiago, N., with Navarro, 779 (Mal.)
Santiago Stevenson, D., with Oliver González, Biaggi, Acevedo, Mariotta, Hernández Morales & Pérez Santiago, 54 (Hel.)
Santos, J. M., with Paraense, 272, 478 (Hel.)

Santos, O. O., with de Bustamante, Lacerda & Paracampos, 319 (Mal.)
Santos Zetina, F., 644 (Lep.)
Sanyal, P. K., 585 (Misc. Dis.)
Sanz, R., with Neghme, Miranda & Agosin, 738 (Am.)
Van der Sar, A., 829 (Def. Dis.)
Sarandria, J. L., with Wertman, 726 (Typh.)
Sardadi, M., with Hill & Kodijat, 1117 (Ys.)
Sarkies, J. W. R., 756, 1043 (Oph.)
——, with Hughes, 1031 (Hel.)
Sarkissian, M., with Lagrange & Scheecqmans 1013 (Hel.)
Sarmiento, A. P., with Pesigan & Pangilinan, 656 (Hel.)
Saroff, A. H., with Huff, Marchbank, Scrimshaw & Shiroishi, 446 (Mal.)
Sasa, M., 411 (Ent.)
——, Kano, R. & Hayashi, S., (293) (Ent.)
——, ——, ——, Kimura, M., Miura, A., Oyama, K., Sato, K., Hosoya, H. & Hasegawa, K., 588 (Ent.)
——, —— & Takahasi, H., (293) (Ent.)
Sasse, B. E., 207 (Ent.)
Satta, E., 628 (Rab.)
Sato, K., with Sasa, Kano, Hayashi, Kimura, Miura, Oyama, Hosoya & Hasegawa, 588 (Ent.)
——, Tadokoro, I., Kurihara, T. & Ibuka, K., 987 (Chl.)
Sato, M., with Hashimoto, Adachi & Hasegawa, 566 (Lep.)
Sato, S., 827 (Def. Dis.)
Satya Prakash, with Raghavan, 137 (Leish.)
Sauer, A. & Weissflug, I., 188 (Hel.)
Saunders, G., 238 (Tryp.)
Sautet, J., Ranque, J., Vague, J. & Vuillet, J., 147 (Am.)
Sawyer, W. A., (601) (Reports, etc.)
Scaffidi, V., (247) (Typh.), 1057 (B.R.)
Scaillet, L. & Haddad, E., 961 (Tryp.)
Schaberg, A., with Straub, 63 (Def. Dis.)
Schaeffer, K., Loughlin, E. H. & Joseph, A., 562 (Ys.)
——, with —— & ——, 560, 562 (Ys.)
Schaible, E., with Meythaler, 1066 (Mal.)
Scharyj, M., with Campins & Cortés, 402 (Der.)
Scheecqmans, G., with Lagrange, 571, 1011, 1012 (Hel.)
——, with —— & Sarkissian, 1013 (Hel.)
Scheikevitch, V., 892 (Am.)
Schiavi, A. & de Mello, V. H., 857 (Mal.)
—— & Proença, L. M., 333 (Mal.)
——, with Wasicky & Unti, 20 (Mal.)
Schiller, E. L., with Rausch, 661 (Hel.)
Schinazi, L. A., Drell, W., Ball, G. H. & Dunn, M. S., 131 (Mal.)
Schisani, J. O., with Carri, 737 (Am.)
Schlesinger, P. & Benchimol, A. B., 1139 (Def. Dis.)
Schlosser, R. J., 59 (Hel.)
Schmidt, B. & Wieland, F., 176 (Hel.)
Schmidt, F. R., 243 (Leish.)
Schmidt, J. & Mendheim, H., 183 (Hel.)
—— & ——, with Hannak, J., 577 (Hel.)
——, Schneid, G. & Mendheim, H., 62 (Hel.)
Schneid, G., with Schmidt & Mendheim, 62 (Hel.)
Schneider, H.-H., with Lendle, 912 (Hel.)
Schneider, J., 612 (Mal.), 650 (Lep.), 736 (Am.)
——, with Laviron & Lauret, 1124 (Lep.)
——, Laviron, P., Lauret, L. & Basset, A., 651 (Lep.)
—— & Montézin, G., 265 (Am.)
—— & Rayroux, J., 268 (Lep.)
Schneider, R. G. & Levin, W. C., 192 (Haem.)
Schnitzer, R. J., with Grunberg, 1001 (Lep.)
Schoeman, J. J., 1060 (Mal.)
Schoenaers, F., (762) (Ent.)
—— & Gregoire, C., (765) (Reports, etc.)
van Schoor, A., with Zima, v. Werder, Hoffmann & Hepding, 186 (Hel.)
Schroeder, H. O., with Jones & Incho, 211 (Ent.)
Schubert, J. H., with Hill, Morlan & Utterback, 976 (Typh.)
—— & Kelley, M. J., 432 (Mal.)
——, Stanford, S. M. & Tiffany, E. J., 624 (Typh.)
Schubert, M., 380 (Hel.)
Schubothe, H., with Heilmeyer & Müller, 831 (Haem.)
Schüffner, W., 909 (Hel.)
—— & Bool, J., 908 (Hel.)
——, Swellengrebel, N. H. & Bool, J., 61 (Hel.)
Schultz, W., with Westphal, 408 (Prot.)
Schulz, K. H., 36 (Pl.)
Schwardt, H. H., with Pimentel & Norton, 413, 680 (Ent.)
Schwartz, R., Mansuy, M. M. & John, W. C. 437 (Mal.)
Schwarz, J. & Muth, J., (497) (Der.)
Schwendy, J., 828 (Def. Dis.)
Schwetz, J., 481, 655 *bis*, 1002, 1003, 1004 *bis*, 1007, 1011, (1014) (Hel.), 511 (Mal.), 591 (Ent.)
——, with Baumann, H. & Fort, M., (745), (1014) (Hel.), 855 (Mal.)
Schwink, T. M., with Becker, 527 (Mal.)
——, with —— & Brodine, 1084 (Mal.)
——, with —— & Prather, 1085 (Mal.)
Scott, J. A., with Faust & Taylor, 108 (Mal.)
Scott, O. K., with Huntington & Eichold, 488 (Hel.)
Scott, V., 123 (Mal.)
Scrimshaw, N. S., Guzman, M. & Mendez de la Vega, J., 827 (Def. Dis.)
Scrimshaw, P. W., with Huff, Marchbank, Saroff & Shiroishi, 446 (Mal.)
Seal, S. C., 861 (Mal.), 985 (Pl.)
—— & Mukherji, S. P., 985 (Pl.)
Sebaoun, with Siguier, Choubrac & Piette, 808 (Am.)
Seife, M. & Lisa, J. R., 168 (Hel.)
Seiser, A., 567 (Hel.)
Selwyn-Clarke, S., 763 (Reports, etc.)
Sen, N., with Dharmendra & Chatterji, 164 (Lep.)
Sen, P., (10) (Mal.)
Seneca, H. & Henderson, E., 359 (Chl.), 1092 (Tryp.)
Sen Gupta, P. C., 25, 721 (Leish.)
——, with Chakravarti, 350 (Leish.)
——, with Chakravarty, Bose & De, 136 (Leish.)
——, with Das, 350 (Leish.)
——, Mallick, K. C. B. & Bhattacharya, B., 594 (Lab.)
——, Panja, D. & Banerjee, A. K., 454 (Leish.)
Sen Gupta, P. N., 889 (Chl.)
Sen Gupta, R. K., with Subramanian, 433 (Mal.)
Sepulveda, G., Jr. & Ibarra, L. M., 739 (Ys.)
Sergent, E., 920 (Vms.)

Sergent, Ed., 317 (Mal.)
—— & Parrot, L., 516 (Mal.)
—— & Poncet, A., 445 (Mal.)
Serrano, J. M., with Rehn & Henderson, 113 (Mal.)
Servino, V., 926 (Ulc.)
—— & Tarizzo, M., 583 (Ulc.)
Sessoms, S. M., with Terry & Lewis, 535 (Leish.)
Seuc-Chiu, A., with Basnuevo, 382 (Hel.)
Shaby, J. A., with Al-Witry & Kantarjian, 750 (Def. Dis.)
Shaffer, J. G. & Biegeleisen, J. Z., 734 (Am.)
——, with ——, 734
Shah, I. A., with Rafi, 1071 (Mal.)
Shah, M. T., with Naik & Nargund, (566) (Lep.)
Shannon, J. A., 707 (Mal.)
Shapiro, M., (917) (Haem.)
Sharif, M., 983 (Pl.)
Sharma, M. I. D., with Dakshinamurty, 676 (Ent.)
Sharp, T. M., 1006 (Hel.)
Shaw, G. D., with Macaulay, (346) (Tryp.)
Shearer, A. R., with Dolman, Kerr & Chang, 642 (Am.), 994 (R.F.)
Shelmire, B., with Lamb, Cooper, Morgan & Keaty, 71 (Der.)
Sheris, E., with Hengel & Kausche, 249 (Typh.)
Shibuki, M., 1104 (Rab.)
Shiroishi, T., with Huff, Marchbank, Saroff & Scrimshaw, 446 (Mal.)
Shishido, A., with Kitaoka, 624, 973 (Typh.)
Shively, J. A. & Kuhns, D. M., 162 (Lep.)
Shookhoff, H. B., 1113 (Am.)
Shortt, H. E., 110 (Mal.), 583 (Misc. Dis.)
——, Fairley, N. H., Covell, G., Shute, P. G. & Garnham, P. C. C., 701 (Mal.)
Shotton, D., Crockett, C. L., Jr. & Leavell, B. S., 753 (Haem.)
Shuman, B., with Israels, 205 (Ent.)
Shute, P. G. & Maryon, M., 702 (Mal.)
——, with Shortt, Fairley, Covell & Garnham, 701 (Mal.)
Sicé, A. & Binder, T., 780 (Mal.)
——, with Joyeux, 302 (B.R.)
Sicuteri, F., 799 (Typh.)
Sidky, M. M., 29 (Typh.)
—— & Freyche, M. J., 75 (Oph.)
Sieniawski, J., with Janssens & Verstraete, 864 (Mal.)
Signorini, F. L. & Terni. M. 44 (Lep.)
——, with ——, 266, 642 (Lep.)
Signorini, L. F., with Davoli, 800 *bis* (Typh.)
——, with Del Turco & Davoli, 799 (Typh.)
——, with Ridi, —— & Ceccherini, 799 (Typh.)
——, with Simondi & ——, 799 (Typh.)
Siguier, F., with Blanc, 217 (B.R.)
——, Choubrac, Piette, M. & Herman, J., 468 (Am.)
——, ——, —— & Sebaoun, 808 (Am.)
——, Piette, M., Choubrac, P. & Herman, 155 (Am.)
——, —— & Crosnier, J., 38 (Am.)
Si Hassen, A., with Méchin, 723 (Leish.)
Siim, J. C., with Christiansen, 1150 (Prot.)
Sikes, R. K., with Chamberlain, 293 (Ent.)
da Silva, A. C., 600 (Reports, etc.)
D'Silva, C. B., 804 (Rab.)
de Silva, C. C. & Weeratunge, C. E. S., 1038 (Haem.)
Silva, F., 559 (Ys.)
Da Silva, J. M., with Pires & Teles E Cunha, 448 (Tryp.)
da Silva, L. S., 150 (Am.)
da Silva, O. J. & Forattini, O. P., 793 (Tryp.)
Silva, R., with Neghme, 155 (Am.)
——, with ——, Bertín, Donckaster, Artigas, Bunster, Munoz T., Faúndes & Basso, 484 (Hel.)
——, with ——, ——, Tagle & Artigas, 484 (Hel.)
da Silva, T. L., 835 (Vms.)
Silva Goytia, R., 356 *bis* (Typh.)
Silver, H. K., 280 (Haem.)
Simmons, S. W., 214 (Reports, etc.)
—— & Upholt, W. M., 782 (Mal.), (1157) (Ent.)
Simon, A., with Lichtenstein, 657 (Hel.)
Simondi, U., Davoli, R. & Signorini, L. F., 799 (Typh.)
Simonson, E., with Keys, Brožek, Henschel, Mickelsen, Taylor, Skinner & Wells, 767 (B.R.)
Simpson, T. W., with Mackie, Tuttle & Kluttz, 857 (Mal.)
Siniscal, A. A., 286 (Oph.)
Sitnitskaja, I. G., 611 (Mal.)
Siung, O. H., 279 (Hel.)
Sivalingam, V., with Crusz, 667 (Hel.
Sivaratnam, C., (907) (Hel.)
Skerrett, E. J., Stringer, A. & Woodcock, D., (932) (Ent.)
Skinner, A. S., with Keys, Brożek, Henschel, Mickelsen, Taylor, Simonson & Wells, 767 (B.R.)
Slavin, G., (542) (Typh.)
Sleisenger, M. H., Murray, E. S. & Cohen, S., 728 (Typh.)
Slim Villegas, V. J. & Aranda Reyes, B., (497) (Der.)
Sloan, N. R., with Arnold, 998 (Lep.)
Smadel, J. E., with Jackson, 795, 978 (Typh.)
——, Jackson, E. B. & Ley, H. L., Jr., 978 (Typh.)
——, Ley, H. L., Jr., Diercks, F. H. & Traub, R., 355 (Typh.)
——, ——, ——, ——, Tripton, V. J. & Frick, L. P., 977 (Typh.)
Smith, C. C., 441 (Mal.)
Smith, C. E., Saito, M. T., Beard, R. R., Kepp, R. McF., Clark, R. W. & Eddie, B. U., 73 (Der.)
Smith, C. N., with Travis, 932 (Ent.)
Smith, D. A., 390 (Def. Dis.)
Smith, H. F. & Dy, F. J., 18 (Mal.)
——, —— & Del Rosario, F. G., 112 (Mal.)
Smith, M., with Ramanujam, 374 (Lep.)
Smith, N., with Blanton, Travis & Husman, 91 (Ent.)
——, with Travis & Applewhite, 91 (Ent.)
Smith, P., with Garber & Wolochow, 805 (Pl.)
Smith, R. G., with Philpot, 69 (Vms.)
Smith, W. C., with Salis, 570 (Hel.)
Smyth, J. D., (1020) (Hel.)
Snow, W. E., with Ludvik & Hawkins, 616 (Mal.)
Snyder, J. C., with Murray, 536 (Typh.)
Snyder, M. J., with Bauer, Parker, Hall, Benson, Joslin, Hightower, Venable & Woodward, 979 (Typh.)
——, with Powell, Minor, Benson & Woodward, 979 (Typh.)
Soap, 211 (Ent.)
Sofia, F., 215 (Reports, etc.)
Sohier, H., Paris, P. & Camain, R., (1023) (Hel.)
Sokal, J. E., 121 (Mal.)

Sokhey, S. S. & Habbu, M. K., 256 *ter.* (Chl.)
——, —— & Bharucha, K. H., 256 (Chl.)
de Soldati, L. & Rabenko, J., 878 (Tryp.)
Sollod, N., with Dagradi & Friedlander, (579) (Haem.)
Soman, D. W., with Felsenfeld, Ishihara, Waters & Norsen, 988 (Chl.)
——, with ——, Young, Yoshimura, Waters & Ishihara, 987 (Chl.)
van Someren, E. C. C., 760 (Ent.)
——, with Haddow, (293) (Ent.)
van Someren, V. D., & McMahon, J., 181 (Hel.)
Sommerville, T., with Mulligan & Lloyd, 21 (Mal.)
Soper, F. L., 758 (Oph.)
Sorour, O., Guirguis, S. & Halim, S. A., (745) (Hel.)
Sotolongo, F., with Basnuevo, Cowley Chávez, Blanco Rabassa & Achkar, 1137 (Hel.)
Soulage, J., Farinaud, M. E., Tauzin, M. & Lefebvre, E., 253 (Pl.)
Soule, M. H., (997) (Lep.)
de Sousa, E. F., with Penido & Bezerra, 235 (Mal.)
de Sousa, J., with Rachou, 411 (Ent.)
de Souza, L. A., with Gomes, 370 (Lep.)
de Souza-Araujo, H. C., 46, 474, 647 *bis* (Lep.)
de Souza Campos, N., (373) (Lep.)
Souza Campos, N., Rosemberg, J. & Aun, J. N., 372 (Lep.)
——, with —— & ——, 373, 1121 *bis* (Lep.)
Souza Lima, J. S., 820 (Hel.)
de Souza Lima, M., with Rath de Souza, 45 (Lep.)
Spät, W., 536 (Typh.)
Spies, T. D., Garcia Lopez, G., Milanes, F., Lopez Toca, R. & Reboredo, A., 400 (Haem.)
——, ——, ——, ——, —— & Stone, R. E. 399 (Haem.)
Spingarn, C. L. & Edelman, M. H., 260, 733 (Am.)
Spindler, L. A., with Harrington & Hill, 280 (Hel.)
Spinks, A., (25), 345 (Tryp.)
——, with Francis, 268 (Lep.)
Sprent, J. F. A., 823 (Hel.)
Srinivasachar, S., with Veliath, 1141 (Def. Dis.)
Srivastava, R. S., 1075 (Mal.)
Stammers, F. M. G., 70 (Vms.)
Standen, O. D., 901, 1006 (Hel.)
Stanford, S. M., with Schubert & Tiffany, 624 (Typh.)
Stanton, M. F., Laskowski, L. & Pinkerton, H., 143 (Bart.)
——, with —— & ——, 730 (Bart.)
Stanyon, J. H., 914 (Hel.)
Starr, D. F., Ferguson, P. & Salmon, T. N., 211 (Ent.)
Stauber, L. A., with Trager & Harel, 790 (Mal.)
van Steenis, P. B., with Kouwenaar & Winckel 938 (B.R.)
di Stefano, R., with Dassen, 371 (Lep.)
Steiniger, F., 43 (Lep.), 111 (Mal.)
Steinitz, H., with Koch, 1130 (Hel.)
Stempien, S. J., Marcus, D. M. & Kelso, R. W., Jr., 635 (Am.)
Stephens, F. F., with Bower & Wibberley, (240) (Tryp.)
Stephens, F. F. & Wibberley, D. G., (240) (Tryp.)
Stewart, F. H. & Badir, G., 498 (Oph.)
Stewart, G. E. S., with Corkill & Creditor, 390 (Def. Dis.)
Stigliani, R., 569 (Hel.)
Stirewalt, M. A., 1008 (Hel.)
——, with Kuntz, 273 (Hel.)
Stoenner, H. G., 626 (Typh.)
——, with Luoto & Huebner, 546 (Typh.)
Stoker, M. G. P., 249 (Typh.)
——, with Coombs, 886 (Typh.)
——, with Marmion, 248 (Typh.)
Stokes, J., 356 (Typh.)
Stone, P. C., with Laske, Howell, Dahm & Cuff, (590) (Ent.)
Stone, R. E., with Spies, Garcia Lopez, Milanes, Lopez Toca & Reboredo, 399 (Haem.)
Storey, N. E., with Kermack, (864) (Mal.)
Straub, M. & Schaberg, A., 63 (Def. Dis.)
Straus, B. & Gennis, J., 614 (Mal.)
Strauss, A., with Carvalho, Braga, Arminante, Altenhein, Rocha & Vieira, 1136 (Hel.)
Strenzke, K., with Mayer, 1053 (Ent.)
Stringer, A., with Skerrett & Woodcock, (932) (Ent.)
Ström, J., 1149 (Prot.)
Struve, E. E., 553 (Am.)
Suarez, R. M., Martinez, E. C., Suarez, R. M., Jr. & Elias Latorre, A., 916 (Sp.)
Suarez, R. M., with Meyer, Suarez & Benitez Gautier, 916 (Sp.)
Suarez, R. M., Jr., with Meyer, Suarez, Martinez & Benitez Gautier, 916 (Sp.)
——, with Suarez, Martinez & Elias Latorre, 916 (Sp.)
Subramanian, R. & Sen Gupta, R. K., 433 (Mal.)
——, Vaid, B. K. & Dixit, D. T., 126 (Mal.)
Sulman, F. G., with Fischl, 34 (Rab.)
Sumerford, W. T., with Jensen & Fay, 416 (Ent.)
Surewicz, W., 388 (Hel.)
Sutliff, W. D., Dulaney, A. D. & Davis, W. L., 11 (Mal.)
Sutton, D., 1047 (Prot.)
Swamy, T. V., (28) (Typh.)
Swanson, L. E., with Batte & Murphy, 572 (Hel.)
Swellengrebel, N. H., 146 (Pl.), 229, 781 (Mal.)
——, with Bonne-Wepster, 128 (Mal.)
——, with Schüffner & Bool, 61 (Hel.)
Swerts, L., with Dubois, 741 (Lep.)
Symmers, W. St. C., 748 (Hel.), 999 (Lep.)

T

Tabbara, R., with Kazzaz, 1132 (Hel.)
Tabone, V., 1042 (Oph.)
Tabosa, W., with Deane, Freire & Ledo, 19 (Mal.)
Tabusse, with Duroux & Marty, (274) (Hel.)
Tadokoro, I., with Sato, Kurihara & Ibuka, 987 (Chl.)
Tagle, I., with Neghme, Bertín, Silva & Artigas, 484 (Hel.)
Taha, A., with Halawani, Latif & Khalaf, 1013 (Hel.)
Taj-el-Din, H. M., with Jalili & Nakkash, 751 (Haem.)
Takada, S., with Inoki, Nagai, Kitamura & Nakabayashi, 633 (Am.)
Takahasi, H., with Sasa & Kano, (293) (Ent.)
Takeda, S., 391 (Def. Dis.)
Talaat, S., with Erfan, 49 (Hel.)
Talice, R. V., 99 (Reports, etc.)
Tamalet, J., with Paillas, (745) (Hel.)
Tamemasa, O., with Asano, Kameda, Ishiii & Chida, 1086 (Mal.)

Tanguy, F., with Auffret, 263 (Am.), 669 (Def. Dis.)
Tanret, P., with Binet & Bochet, (877) (Bl.)
Tarizzo, M., with Servino, 583 (Ulc.)
Tauzin, M., with Soulage, Farinaud & Lefebvre, 253 (Pl.)
Tavares, A., 159 (Ys.)
——, with Ribas, 560 (Ys.)
Taylor, D. J., Greenberg, J., Highman, B. & Coatney, G. R., 639 (Am.)
——, —— & Josephson, 873 (Mal.)
——, ——, Josephson, E. S. & Ray, A. P., 873 (Mal.)
——, with Josephson, Greenberg & Ray, 862 (Mal.)
Taylor, H. L., with Keys, Brožek, Henschel, Mickelsen, Simonson, Skinner & Wells, 767 (B.R.)
Taylor, H. M., 591 (Ent.)
Taylor, J., 546 (Typh.)
Taylor, J. A. & Riddell, W. A., 377 (Hel.)
Taylor, J. E., with Faust & Scott, 108 (Mal.)
Teesdale, C., 411 (Ent.)
Teir, H., with Telkkä, Putkonen & Hortling, 740 (Lep.)
Telcharov, L. & Todorowa, M., 517 (Mal.)
Teles e Cunha, G., with Pires & Da Silva, 448 (Tryp.)
Telkkä, A., Putkonen, T., Teir, H. & Hortling, H., 740 (Lep.)
Ten Berg, J. A. G., 167, 656 (Hel.)
Tenbroeck, C., 458 (Rab.)
—— & Herriott, R. M., 458 (Rab.)
Tendeiro, J., 881 (Typh.)
——, with Ferreira, 241 (Tryp.)
Teotia, T. P. S. & Dahm, P. A., 762 (Ent.)
Termine, L., with d'Ignazio, 353 (Typh.), (365) (Am.)
Terni, M. & Signorini, F. L., 266, 642 (Lep.)
——, with ——, 44 (Lep.)
Terry, L. L., Lewis, J. L., Jr. & Sessoms, S. M., 535 (Leish.)
Terzian, L. A., 447 (Mal.)
Teusch, W., with Wolff, 173 (Hel.)
Tew, R. P., David, W. A. L. & Busvine, J. R., 589 (Ent.)
Théodoridès, J., (93) (Ent.)
Thiel, C. C., with Marmion, MacCallum, Rowlands, McDonald & Phipps, 1100 (Typh.)
van Thiel, P. H., 88 (Prot.)
Thillet, C. J., with Oliver González, 53 (Hel.)
Thiodet, J., 120 (Mal.), 903 (Hel.)
——, Butori, P., Fourrier, A. & Massonnat, J., 468 (Am.)
——, Pietri, H., Fourrier, A., Massonnat, J. & Aubry, P., 992 (Am.)
Thomas, A., (713) (Mal.)
Thomas, I. M., 205 (Ent.)
Thompson, B. C., 583 (Misc. Dis.)
Thompson, P. E., with Reinertson, 1113 (Am.)
Thompson, T., 546 (Typh.)
Thomson, F. A., 395 (Def. Dis.)
Thomson, R. C. M., see Muirhead-Thomson, R. C.
Thraikill, F. B., with Parker, Pickens, Lackman & Bell, 796 (Typh.)
Thurman, D. C., Jr. & Mortenson, E. W., 677 (Ent.)
——, with Peters, Markos & Mulhern, 91 (Ent.)
Thurston, J. P., 21, 869 (Mal.)
——, with Hawking, 868 (Mal.), 1088 (Tryp.)
Tiant, F. R., with Pardo-Castello, Trespalacios, Ibarra, Jose Mestre & Beade, 652 (Lep.)
Tierkel, E. S., Graves, L. M., Tuggle, H. G. & Wadley, S. L., 35 (Rab.)
Tiffany, E. J., with Schubert & Stanford, 624 (Typh.)
Timtschinov, N., 936 (Reports, etc.)
Tipton, V. J., with Smadel, Ley, Diercks, Traub & Frick, 977 (Typh.)
Tisseuil, J., 278 (Hel.), (815) (Lep.)
Titiz, I. & Erkip, H., 30 (Typh.)
Tobback, L., (964) (Tryp.)
Tobie, E. J., with von Brand & Mehlman, 530, 964 (Tryp.)
Tobie, J. E., Most, H., Reardon, L. V. & Bozicevich, J., 1110 (Am.)
Todorowa, M., with Telcharov, 517 (Mal.)
Tolentino, J. G., 163, 742 (Lep.)
Tolentino, P., (830) (Haem.)
Tomás Aparisi, D., 816 (Lep.)
Tomlinson, T. H., Jr., with Wilcox & Ballard, 131 (Mal.)
Tonkin, I. M., with Baird, 883 (Typh.)
Torii, S., 392 (Def. Dis.)
v. Törne, H., with Piekarski, 500 (Prot.)
Torres, J. M., with Rodriguex-Molina, Acevedo, Lopez-Sanabria & Ramirez-Rodriguez, 481 (Hel.)
Torrico, J. R., with Torrico, (719) (Tryp.)
Torrico M., R. A., 136 (Tryp.), 738 (Am.), 818, 826 (Hel.)
—— & Torrico, J. R., (719) (Tryp.)
Toulet, J., with Hillemand & Gilbrin, (275) (Hel.)
Toumanoff, C., 89 *bis* (Ent.)
Toušek, M., 559 (R.F.)
Trager, W., 130 (Mal.)
——, Stauber, L. A. & Harel, S. B., (Mal.)
Tran Anh, with Huard, 666, 1030 (Hel.)
Trapido, H., with Fairchild, 680 (Ent.)
——, with Galindo & Carpenter, 144, 802 (Y.F.)
Traub, R., with Smadel, Ley & Diercks, 355 (Typh.)
——, with ——, ——, ——, Tipton & Frick, 977 (Typh.)
Travassos, J., with Guimaraes, 369 (Ys.)
——, with Pereira & Cardoso, 979 (Typh.)
de Traverse, P. M., with Claisse, di Matteo & Deuil, 970 (Leish.)
Travis, B. V., 296 (Ent.)
——, Applewhite, K. H. & Smith, N., 91 (Ent.)
——, with Blanton, Smith & Husman, 91 (Ent.)
—— & Smith, C. N., 932 (Ent.)
Trawiński, A., 167 (Hel.)
Tredre, R. F., 940 (B.R.)
Trembley, H. L., with Greenberg & Coatney, 620 (Mal.)
Trespalacios, F., with Pardo-Castello, Tiant, Ibarra, Jose Mestre & Beade, 652 (Lep.)
Treviño, A., with Colorado Iris, Domínguez & Mazzotti, 55 (Hel.)
Tribedi, B. P. & Roy, A. R., (990) (Am.)
Trincão, C. & de Gouveia, E., 239, 792 (Tryp.)
——, Pinto, A. R., de Almeida, C. L. & Gouveia, E., 281 (Haem.)
Trinidad Govt., 8 (Mal.)
Tripodi, P., 376 (Hel.), 465 (Am.)
——, with Lippi, 242 (Leish.)

Troescher, C. B., with Munsell, Williams, Guild, Nightingale & Harris, (751) (Def. Dis.)
Troitskij, S. A., with Grennaus & Miloradovskaja, 611 (Mal.)
Trub, C. L. P. & Gaase, A., 913 (Hel.)
Truhaut, R., 1052 (Ent.)
Tsai, K. C., with Koenigstein & Chiu, 533 (Leish.)
Tsao, Y., with Chung, Chang, Chang, Feng & Lu, 242 (Leish.)
Tsopellas, V., 286 (Oph.)
Tsuchiya, H. & Aitken, L. F., (176) (Hel.)
—— & McCoy, J. E., 860 (Mal.)
Tucker, H. A., 572 (Hel.)
——, with Hoekenga, 285 (Der.)
Tuckwell, E. G., 81 (Misc. Dis.)
Tuggle, H. G., with Tierkel, Graves & Wadley, 35 (Rab.)
Del Turco, L. R., Davoli, R. & Signorini, L. F., 799 (Typh.)
Turrell, E. S., with Robinson, Gerking & Kincaid, 78 (Heat Str.)
Tuttle, R. L., with Mackie, Simpson & Kluttz, 857 (Mal.)
Tuxen, S. L., 204 (Ent.)
Tveit, J., with Böe & Lindén, 754 (Der.)
Twinn, C. R., 92 (Ent.)
——, with Hocking & McDuffie, 208 (Ent.)
——, with —— & Richards, (90) (Ent.)

U

Udenfriend, S., with Brodie, 14 (Mal.)
Uebel, H., 116 (Mal.), 534 (Leish.)
Unti, O., with Wasicky & Schiavi, 20 (Mal.)
Upholt, W. M., with Simmons, 782 (Mal.), (1157) (Ent.)
Upton, A. C., 383 (Hel.)
Urbach, H., with Herzberg, 885 (Typh.)
——, with —— A Herzberg-Kremmer, 544 (Typh.)
Urbino, C. M., with Ejercito, 703 (Mal.)
Uribe, P., with Gajardo Tobar, Apablaza, Benavides, Vargas, Cepeda, Rojas, Zeldis, Lucchini & De la Fuente, 381 (Hel.)
Urso, B., 465 (Am.)
Ustvedt, H. J., 1120 (Lep.)
Utterback, B. C., with Hill, Morlan & Schubert, 976 (Typh.)
Uyguanco, M. L. G., Rodriguez, J. N. & Concepcion, I., 997 (Lep.)
Uzan, M., 737 (Am.)

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Vachon, M., (920), 1038 (Vms.)
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